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CLINICAL RADIOLOGY OF ACUTE ABDOMINAL DISORDERS

Bernard S. Epstein, M.D.

Chief, Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, New York
Associate Clinical Professor of Radiology, Albert Einstein College of Medicine,
Yeshiva University, New York, New York.

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ALICE, RICK and JONSIE

Preface

THIS work started with the thought of concisely correlating the radiologic manifestations of the "acute abdomen" with clinical and pathological processes. At the outset it became evident that the definition of "acute abdomen" was quite indefinite. Some held that the condition referred only to illness of a dramatic and catastrophic nature. Others preferred a broader usage, regarding any abdominal disturbance requiring medical attention as a condition which, while less intense, nevertheless was acute. It became apparent that the term could not be restricted to a single group of symptoms or diseases, and that one could not state clearly just how or when an abdominal disorder became an "acute abdomen."

Thus, from a rather narrow consideration we came to the broader aspect of acute abdominal disorders. The diversity of clinical symptoms, if one reflects for a moment, can be reduced to a list of well-known manifestations such as pain, nausea, vomiting, distension, constipation, diarrhea and the other basic symptoms. It is apparent that the eliciting of a picture rather than of a list of complaints is fundamentally important in arriving at a diagnosis. Another moment's reflection will show that qualifying adjectives and adverbs, a sense of timing of symptoms, and careful correlation of complaints with the observations made on physical examination may reveal a condition which is not acute insofar as drama is concerned, but nevertheless is one which requires prompt medical attention. In this way, abdominal complaints which are not those of an "acute abdomen" still may indicate an urgent need for help.

At this point the radiologist has a vital position in the development of the diagnosis and the ultimate course of treatment. It is within his realm that the shadows of disease are reflected in gray, black and white. I have not attempted to cover radiologic technic, presuming that anyone attempting abdominal examination is well grounded in such fundamentals. Nor will I assess the relative value of one view against another, or of fluoroscopic examination as apposed to films. It is impossible to generalize, except to say that an examination poorly conceived and executed even with the most modern equipment, and inaccurately interpreted with the best of secretarial gadgetry, still jeopardizes the patient's well-being. Hence, the saving of films, or reliance on fluoroscopic examination alone, or the performance of the examination by unskilled and untrained professional or technical persons is to be deplored. By the same token, and this is particularly true in children and young adults, every effort must be made to keep exposure to radiation to a minimum. This, then, is the province of the radiologist or anyone using radiologic methods—to help, not to harm; to be thorough, not to relegate responsibility to technicians or unqualified assistants; to assume the privileged responsibility of proper examination while the patient is being examined, not to wait until tomorrow morning to "read the films." In short, to render a medically trustworthy and valuable consultation is the purpose of the examination. There is no such thing as "routine," and the most dangerous study is the one reported as "normal."

The text has been purposely kept to a minimum, and references to the literature are scant. Appended to each section is a list of articles worth reviewing as a start towards more detailed information. Imbedded in the text are the observations of many, many workers, and my thanks goes to them and to my teachers, colleagues, friends and house officers who contributed in the slow accumulation of the material. Almost all the illustrations are from my own experience. The others are acknowledged with thanks in the legends. The material upon which this work is based was seen mainly at the Jewish Hospital of Brooklyn and the Long Island Jewish Hospital. I take pleasure in acknowledging the many pleasant associations I had with the former, and now have with the latter. I am grateful to Mr. Jerome Sloven, my good friend and chief technician for many years, for his devoted attention in furthering this work. The photographs were made by Mr. Marvin Ehlin. My secretary, Mrs. Ann Duncan was assisted by Mrs. Jarmila Dvorak. To all of them, my thanks.

The Lea & Febiger organization has been most considerate and helpful, and I am indebted to them for their whole-hearted cooperation. It is indeed a privilege and a pleasure to work with such pleasant and competent people.

BERNARD S. EPSTEIN

New Hyde Park, N.Y.

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Chapter 1

Congenital Disorders

EMBRYOLOGIC CONSIDERATIONS

Man's propensity for gastrointestinal disturbances begins at about the fifteenth day of embryonal life, when the primitive digestive tube consists of the foregut within the cephalic flexure, the hindgut in the caudal flexure, and the open yolk sac between. The cloaca, which is the terminus of the hindgut, extends backwards into the body stalk as the tube of the allantois. As the yolk sac diminishes, its neck is reduced to a narrow tube termed the yolk stalk or vitelline duct. The anterior end of the foregut is separated from the stomodeum by the buccopharyngeal membrane. The upper foregut later dilates to form the pharynx, where the branchial arches develop. The esophagus forms behind this and its elongation follows the development and descent of the stomach, which begins at about the fourth week of fetal life. Caudal to this the gut opens freely into the yolk sac. The liver forms as a diverticulum between the stomach and the mouth of the vitelline duct. From the stomach to the rectum a mesodermal band appears which develops into the mesentery, fixing the primitive gut to the notochord. The stomach becomes attached to the ventral abdominal wall as far as the umbilicus by the septum transversum. The cephalic portion of this septum helps form the diaphragm, while the caudal aspect, into which the liver grows, forms the ventral mesogastrium. As the stomach dilates the greater curvature is directed towards the spine, while the lesser curvature is directed ventrally.

At about the sixth week the midgut becomes elongated into a V shaped loop which projects downwards and forward. At the apex of the V the vitelline duct passes into the umbilicus. The buds of the spleen and pancreas appear behind the stomach, the former above the latter. From the aorta the celiac artery extends to supply the spleen, pancreas and stomach. The superior mesenteric artery fits into the V loop of the midgut, and the inferior mesenteric artery supplies the hindgut. All lie in a common posterior mesentery, above which is the dorsal mesogastrium attached to the greater curvature of the stomach. The dorsal mesentery suspending the colon is the mesocolon. At about this time another local enlargement appears in the gut just behind the opening of the vitelline duct. This forms the cecum and, later, the appendix. The loop of gut distal to the cecal diverticulum becomes the ascending and transverse colon. The hindgut, which bulges into the cloaca, divides into a tubular rectum dorsally directed to the anus, and a ventral urogenital sinus. Thus, at 6 weeks the digestive tract is a continuous tube from the pharynx to the anus. By the end of the third month the midgut is withdrawn from the umbilical cord and returns to the abdominal cavity.

In the seventh to tenth week the stomach grows rapidly. The greater curvature attached to the dorsal mesogastrium grows faster than the lesser curvature and its attached ventral mesogastrium. The greater curvature rotates downward and toward the left, so that its right surface turns posteriorly and its left anteriorly. The dorsal mesogastrium, following the rotation of the greater curvature becomes elongated, and is drawn away from the spine laterally and ventrally. With this rotation, the right surfaces of both the ventral and dorsal mesogastriums become directed backwards, and the left side of the stomach becomes directed forward. Thus the omental bursa is formed behind the stomach, and this enlarges as the gastrointestinal tract grows. The entrance to the lesser peritoneal sac is the foramen of Winslow (foramen epiploicum). Because of the bulging of the greater curvature, the caudal end of the stomach is pointed ventrally, or

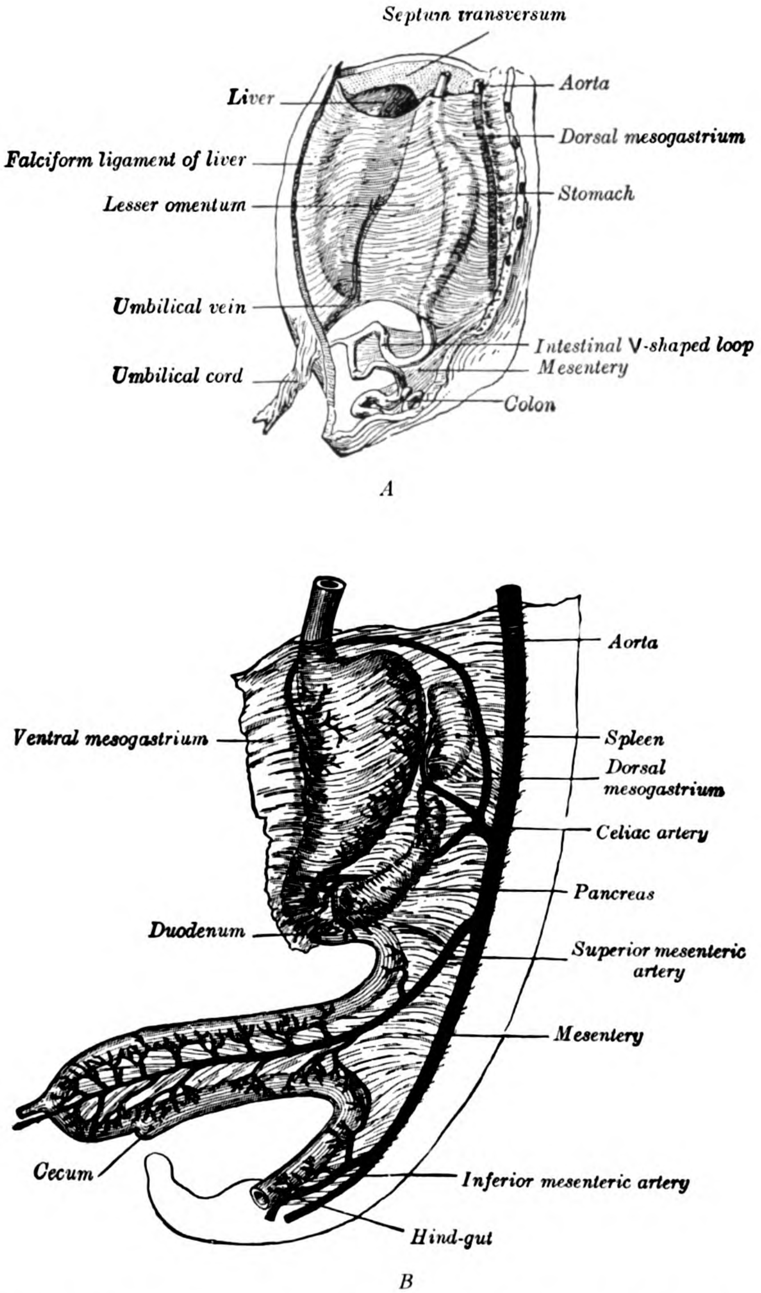

FIG. 1.—*A*. The primitive mesentery of a six weeks' human embryo, half schematic. (Kollmann in *Gray's Anatomy*.)

B. Abdominal part of digestive tube and its attachment to the primitive or common mesentery. Human embryo of six weeks. (After Toldt in *Gray's Anatomy*.)

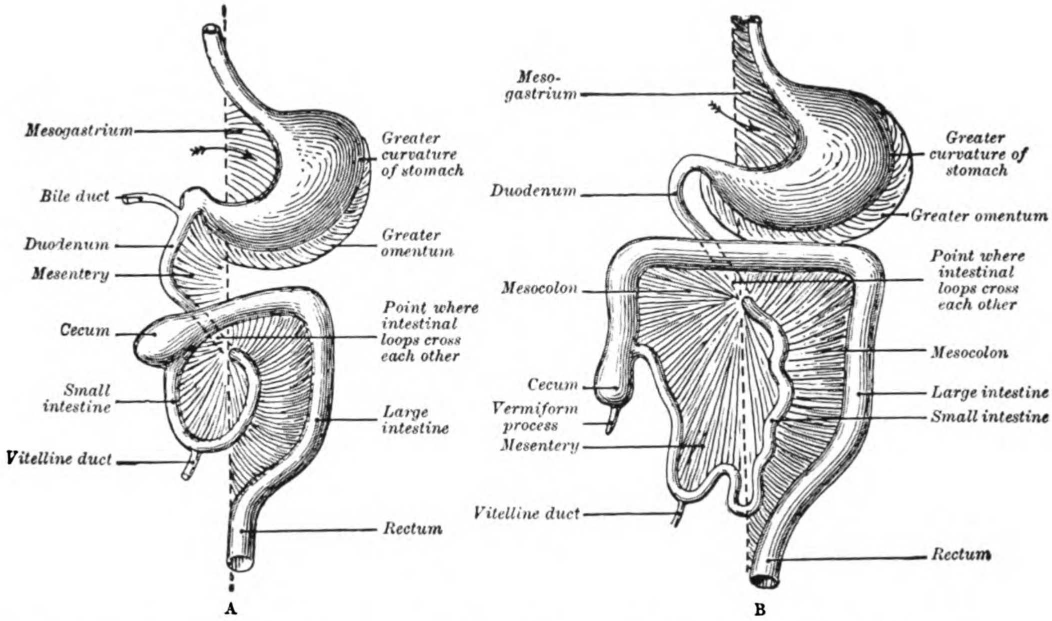

FIG. 2.—Diagrams to illustrate two stages in the development of the digestive tube and its mesentery. The arrow indicates the entrance to the bursa omentalis. (*Gray's Anatomy.*)

occasionally caudally. This produces a reverse bend in the duodenum beneath. As the stomach rotates to the right it carries the duodenal sweep in the same direction.

The liver and pancreas arise as diverticula from the duodenum, and being fixed, keep the duodenum in position. The duodenum is suspended by a mesentery at first, and projects forward as a loop. The duodenal mesentery and loop are subsequently displaced by the transverse colon. The right surface of the mesentery is directed posteriorly, and adhering to the parietal peritoneum, gradually disappears. Minor rotational defects at this time may alter the position of the duodenum.

As the stomach rotates the lesser peritoneal sac, or the bursa omentalis, is formed by the elongation of the dorsal mesogastrium. The peritoneum grows downward from the greater curvature in front of the transverse colon and small intestine to form the greater omentum. The lesser omentum is derived from the ventral mesogastrium attached to the anterior abdominal wall, the stomach and duodenum. As the liver grows within this mesentery, the lesser omentum forms between the liver and stomach. The falciform and coronary ligaments between the liver, abdominal wall and diaphragm originate from the same mesodermal sheet. At first the anterior layer of the transverse mesocolon is separated from the posterior layer of the greater omentum. When these two unite, the greater omentum remains suspended from the transverse colon. The mesenteries of the ascending and descending colon disappear as a rule, while that of the small intestine becomes inserted obliquely against the posterior abdominal wall.

The foregut up to the liver bud anteriorly, and the hindgut posteriorly, are maintained in a fixed position by their mesenteric attachments. The midgut, however, having no ventral mesentery, can form coils to accommodate a far greater length of rapidly growing intestine within the abdominal cavity. This fixation of the foregut and hindgut make congenital malpositions uncommon, while the mobility of the midgut renders it quite susceptible to rotational errors.

At the time that the alimentary tract is suspended in the midline by the common dorsal mesentery, three divisions are noted, according to form and blood supply. The uppermost loop, the foregut, is supplied by the celiac axis. The midgut, which forms the umbilical loop, is supplied by the superior mesenteric artery. The lower loop, the hindgut is supplied by the inferior mesenteric artery. The midgut grows most rapidly, so by the end of the fourth week the vitello-intestinal duct has lost its connection with the yolk sac and the umbilical cord. The available intraabdominal space is so diminished by the rapid growth of the liver and the midgut loop that the latter is extruded into the root of the umbilical cord as a temporary physiologic umbilical hernia. At the fifth week the midgut still lies in the sagittal plane, the apex of the loop being represented by the remnant of the vitelline duct and the termination of the superior mesenteric artery. The anterior segment of this loop is termed the prearterial segment, and the posterior the postarterial segment. The lower end of the foregut and the upper end of the hindgut become relatively fixed points from which the midgut loop is suspended. The upper part of the duodenum is deviated to the right by the bulging and unequal growth of the greater curvature of the stomach. It becomes fixed in this position by the thickening and shortening of its mesentery and by outgrowth of the pancreatic bud. A retention band passes from the upper hindgut upwards as a thickening in the mesentery to the region of origin of the superior mesenteric artery. This band does not keep pace in growth with the gut or the remainder of the mesentery, and thus fixes the intestine at the junction of the midgut and the hindgut. These two fixed points are close together, and the growth of the embryo causes further approximation. The narrow isthmus which they form and from which the midgut loop hangs is called the duodeno-colic isthmus. This early fixation of foregut and hindgut loops precludes the possibility of either loop rotating extensively, while the midgut passes through a complicated process of rotation.

Three stages of intestinal rotation are recognized. The first occurs when the midgut loop occupies the umbilical hernia and extends to the time when the loop returns to the abdominal cavity at about the tenth week. The second stage of rotation occupies the time during which rotation and reduction of the midgut into the abdominal cavity takes place, and is completed when the cecum extends to the right loin in the eleventh week. The third stage is from this time until shortly after birth. It is characterized by descent of the cecum and fixation of the cecum and lower duodenum by fusion of their mesenteries with the posterior parietal peritoneum.

The essential features of the first stage of rotation is the turning of the intraumbilical loop from the sagittal to the horizontal plane, so that the prearterial segment lies to the right and the postarterial segment to the left. The danger of failure of the intestine to rotate beyond the first stage is seen in exomphalos. In this condition the embryonic hernia into the umbilical cord persists in part or in full until birth. The hernia is covered by the thin and translucent umbilical cord rather than by skin, as in the usual umbilical hernia.

The second stage of rotation takes place quickly, and is characterized by an orderly return of lesser bowel loops into the abdomen. The proximal limb of the prearterial segment returns first, its coils coming to lie to the right of the superior mesenteric artery. As these coils collect, the hindgut and its mesentery which lie in the midline are displaced to the left, backward and upward. The splenic flexure and descending colon thus assume their normal positions. The cecum and ascending colon are reduced last. As they enter the abdomen and the colon is straightened out, they are deflected to the right. The final result of the second stage is a counter-clockwise rotation of the midgut loop about the axis of the superior mesenteric artery 270 degrees from the sagittal position of the primitive umbilical loop. Thus the intestinal tract occupies the adult position, with the duodenum behind the origin of the superior mesenteric artery and the transverse colon crossing the same point anteriorly.

Rotational derangements in the second stage are classified by Dott as nonrotation, malrotation and reverse rotation. With nonrotation, the midgut loop persists in the embryonic placement of approximately 8 weeks, with the small intestine to the right of the midline and the colon to the left. The duodenum descends to the right of the superior mesenteric artery and is mobile on its mesentery. The terminal ileum passes across the midline to reach the colon in the left side

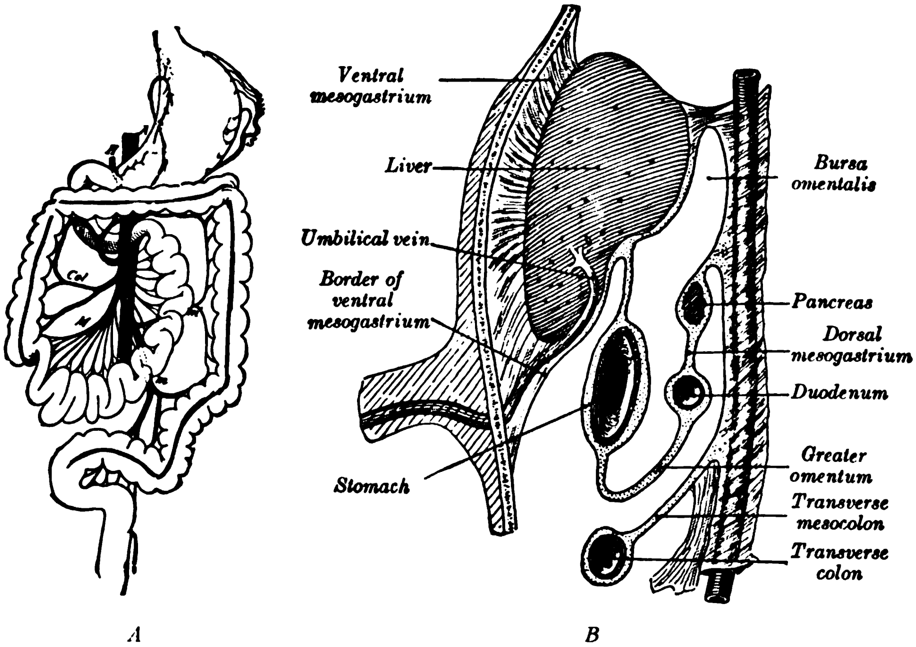

FIG. 3.—*A*, Final disposition of the intestines and their vascular relations (Jonnesco). *A*. Aorta. *H*. Hepatic artery. *M*, *Col*. Branches of superior mesenteric artery. *m*, *m′*. Branches of inferior mesenteric artery. *S*. Splenic artery.

B, Schematic figure of the bursa omentalis, etc. Human embryo of eight weeks (Kollmann). (*Gray's Anatomy.*)

of the abdominal cavity to enter the cecum from right instead of its left side. The ascending colon ascends on the left of the midline to a point behind the greatery curvature of the stomach. The transverse colon is formed by a U-shaped shortened loop. In this position, there may be no secondary fixation of the mesentery, the entire loop of midgut being suspended free from the duodenocolic isthmus. Such an arrangement predisposes to development of volvulus of the entire midgut.

Malrotation is accompanied by irregular defects of rotation and fixation. The cecum and ascending colon may retain their primitive mesentery in common with that of the small intestine (mesenterium commune). The attachment of the root of the mesentery to the abdominal wall is short, and volvulus of the common mesentery can follow. With such abnormal mesenteric fixations there may be some obstruction of the intestinal lumen. The prearterial segment may be reduced in front of the superior mesenteric artery, while the rotation of the postarterial segment is arrested. The postarterial segment may rotate normally while the prearterial segment persists on the right side. Other variations also occur.

With reversed rotation there is a 90 degree clockwise rotation rather than the normal 270 degree counter-clockwise turning. The transverse colon thus lies dorsal to the duodenum and superior mesenteric artery. If the root of the mesentery is fixed towards the right iliac fossa the transverse colon becomes trapped in a tunnel beneath the mesenteric root. Fixation of the cecum and ascending colon is usually incomplete, and torsion of the mobile half of the colon may occur, producing obstruction of the transverse colon at the site of the tunnel through the root of the mesentery.

The third stage of rotation is completed at about the time of birth. The cecum descends and is fixed in its normal position in the right iliac fossa. The descending colon also becomes fixed, as does the lower portion of the

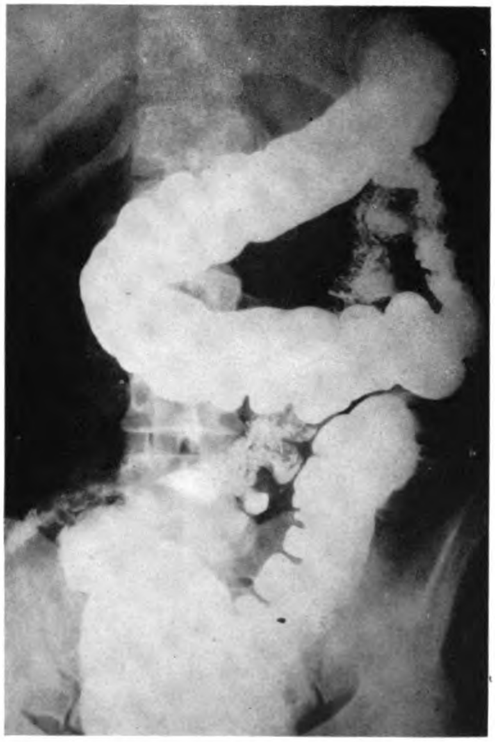

FIG. 4.—Mesenterium commune. Malrotation of the bowel in a 44-year-old woman with the cecum and ascending colon to the left of the midline. The terminal ileum enters the right lateral aspect of the cecum. This had been diagnosed erroneously elsewhere as an obstruction in the midtransverse colon. Diagnosis confirmed at operation for cholelithiasis.

duodenum. The mass of small intestine originally dependent from a narrow pedicle at the origin of the superior mesenteric artery thus acquires a broad oblique attachment to the posterior abdominal wall. Deficiency of this attachment predisposes to volvulus. If the cecum fails to elongate and become properly fixed, it remains in a subhepatic and lumbar position, and is predisposed to volvulus.

By the sixth month the gut has rotated so that the large bowel is situated in front of the small bowel, with the cecum below the liver. At the end of the sixth month the cecum descends to the right lower quadrant, and the colon assumes its ascending, transverse and descending portions. The transverse portion comes to lie in front of the duodenum just below the greater curvature of the stomach.

The changes induced in the position of the common mesentery result in the adult disposition of the mesenteric folds. The greater omentum is formed as the bursa omentalis extends downward from the greater curvature of the stomach in front of the transverse colon and lesser bowel. The continuity between the thoracic and peritoneal cavities is gradually closed, but before this occurs the bursa omentalis sends up a diverticulum on either side of the esophagus. The left one disappears, but the right persists as a small intrathoracic sac to the right of the lower esophagus.

At first the anterior layer of the transverse mesocolon is separated from the posterior layer of the greater omentum. Ultimately these blend, so that the greater omentum appears attached to the transverse colon. The mesenteries of the ascending and descending colon usually disappear. The lesser bowel mesentery then assumes its adult oblique position. The lesser omentum is formed by the thinning ventral mesogastrium which attaches the stomach and duodenum to the anterior abdominal wall. With the growth of the liver this becomes the falciform ligament, and the coronary ligaments between the liver, the abdominal wall and the diaphragm, with the lesser omentum placed between the liver and stomach.

The hindgut first extends backward into the body stalk as the tube of the allantois. With the growth and flexure of the tail end of the embryo, the body stalk is bent forward at the junction of the hindgut and the allantois. A pouch formed at this bend is the entodermal cloaca, and the hindgut opens into its dorsal aspect. The allantois passes forward from its anterior aspect, and later the Wolffian and the Müllerian ducts enter here. The cloaca is shut off by the cloacal membrane formed by the apposition of ectoderm and entoderm. This reaches as far forward as the future umbilicus. Behind the umbilicus the mesoderm later forms the lower abdominal wall and the symphysis pubis. With growth, the cloacal membrane comes to lie at the bottom of a depression lined with ectoderm, the ectodermal cloaca.

A ridge separates the allantois from the cloacal opening. A septum growing down from this ridge into the cloaca divides this cavity into a dorsal anal and a ventral urogenital chamber. These spaces are joined for a time by the cloacal duct, which may persist as an opening between the rectum and the uretha.

The anal canal is formed by an invagination of ectoderm behind the urogenital septum called the proctodeum. When this meets the entoderm of the hindgut it forms the anal membrane. When this membrane is absorbed the rectum and anus are continuous.

The liver arises as a diverticulum from the ventral aspect of the primordial tube which ultimately becomes the descending loop of the duodenum. This diverticulum grows upward and forward into the septum transversum between the vitelline duct and the pericardial cavity. Two buds appear, one for the right and the second for the left lobe of the liver. These buds develop into the hepatic cylinders, which envelope the vitelline and umbilical veins, establishing a series of vascular sinusoids which eventually form the hepatic venous capillaries. With further growth the liver and ventral mesogastrium are gradually differentiated from the septum transversum. The liver projects ventrally into the abdominal cavity, and as it grows the ventral mesogastrium forms the falciform and coronary ligaments from its anterior part and the lesser omentum from its posterior part. By the end of the third month the liver practically fills the abdomen, and the left lobe is equal in size to the right. As growth proceeds, the left lobe development is diminished while the right becomes larger.

The common bile duct is derived from the original diverticulum from the duodenum. The cystic duct and gallbladder arise from this as solid structures which form into hollow organs. The common bile duct originally lies in the anterior duodenal wall, but when the gut rotates it swings into its adult position.

The dorsal aspect of the pancreas arises as a diverticulum from the dorsal duodenum a short distance above the hepatic diverticulum. The pancreas grows upward and backward into the dorsal mesogastrium, forming part of the head and uncinate process as well as the body and tail. The ventral aspect of the pancreas arises as a diverticulum from the primitive bile duct and forms the remainder of the head and uncinate process. These two portions meet and fuse at about the sixth week of fetal life, and the ducts join at this time. The accessory pancreatic duct in the dorsal aspect of the pancreas opens independently into the duodenum. After joining the duodenum it undergoes little or no enlargement. Usually the opening is obliterated, but it may remain patent or persist as a small submucosal nodule. The main pancreatic duct forms in the ventral part of the pancreas and becomes the main passageway opening into the common bile duct.

At first the pancreas lies within the dorsal mesogastrium directed upward and backward, with its surfaces facing right and left respectively. As the stomach changes its position and the dorsal mesogastrium is displaced down and to the left, the right side of the pancreas is directed dorsally and the left ventrally. The right surface becomes applied to the posterior abdominal wall. Its peritoneal coat is absorbed.

The spleen appears as a mesodermal thickening in the dorsal mesogastrium above the tail of the pancreas. With changes in the position of the stomach the spleen is carried to the left, coming to rest behind the stomach in contact with the left kidney. The gastrosplenic ligament is formed by that part of the mesogastrium between the spleen and the greater curvature of the stomach.

In young embryos the thoracic and abdominal cavities are continuous. As the septum transversum develops and separates the heart from the viscera, the ventral part of the diaphragm is formed. The dorsal half of the diaphragm develops from the mesodermal proliferation of the dorsal mesentery. Passages called the pleuro-peritoneal canals remain in the posterolateral aspects of the diaphragm. These are later closed by a membrane formed by the pleura superiorly and the peritoneum inferiorly. Striated muscle then appears between the two serous coats. The foramens of Bochdalek are the remnants of the pleuroperitoneal canals. The foramen of Morgagni remains in the retrosternal area to either side of the midline. If a hernia is formed after the serosal layers of the diaphragm are complete a sac may persist. If

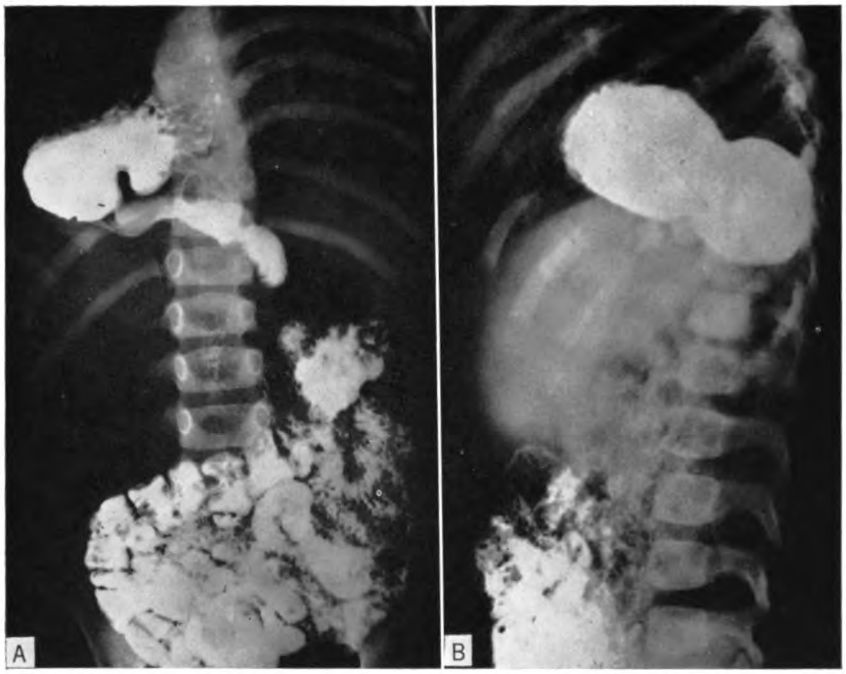

FIG. 5.—*A*, Hernia through the right foramen of Bochdalek in a 2-year-old girl. The stomach is in the posterior aspect of the right hemithorax. This patient also had a double right kidney.
B, Same patient, lateral view.

they are incomplete no sac is present. A sac in Bochdalek herniations is uncommon. With esophageal hiatus hernias sacs are fairly common, and are also seen in about half the cases of foramen of Morgagni hernias.

Additional Reading

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DIAPHRAGMATIC HERNIAS

Congenital defects in the diaphragm occur on either side, and involve the regions of the pleuroperitoneal foramens (foramen of Bochdalek hernias), the dome of the diaphragm in degrees varying from a small perforation to complete absence, or the retrosternal foramens (foramen of Morgagni hernias).

Bochdalek foraminal hernias often manifest themselves soon after birth by dramatic symptoms of dyspnea and cyanosis. The cyanosis varies considerably, becoming more pronounced while the infant is crying or eating. A reluctance to accept food and vomiting often occurs. These symptoms are not specific, occurring whenever abdominal contents enter the chest and displace the lungs, causing pressure atelectasis, and partial obstruction when the intestinal loops rotate.

Hernias through the foramen of Bochdalek are more frequent than other types, occurring on the left side about 5 times more often than on the right. Usually no sac is present. The abdominal contents, often including the spleen, kidney or colon as well as stomach and lesser bowel may extend high into the left pleural cavity, displacing the heart and mediastinum sharply. In the occasional case in which a sac is present, the protruding intrathoracic viscera may be delimited so that the sac simulates the diaphragm.

Right Bochdalek hernias may be associated with displacement of the right lobe of the liver. Sometimes the entire liver enters the right thoracic cavity, displacing the heart and mediastinum to the left.

It is often possible fluoroscopically to identify to some extent the pathologic changes. If the distended bowel contains sufficient gas, the nature of the lesion is evident, particularly on somewhat overexposed films. With pronounced herniations it may be impossible to identify the various loops of intestine without using contrast material, preferably water soluble nonabsorbable media such as those used for intravenous urography. Often the situation is so critical that such investigations are unnecessary, and immediate surgery is required.

Frequently it is not possible to determine with certainty whether a hernia in an infant is through the foramen of Bochdalek or Morgani. Sometimes this can be settled with adequate lateral roentgenograms. However, it is not uncommon to find hernias through the foramen of Bochdalek which extend directly anteriorly, so that the involved portion of the diaphragm cannot be identified. Occasionally, symptoms appear before gas enters the displaced intestinal loops in the thorax. Under such circumstances only a dense opacity is seen, but this becomes altered as soon as gas passes into the intestinal loops.

Other congenital malformations often are seen together with diaphragmatic hernias. The malposition of loops of small and large intestine bespeak the possibility of abnormal mesenteric fixation which predispose to volvulus or obstruction.

Hernias through the *foramens of Morgagni* are less common than pleuroperitoneal canal hernias, the incidence being about 1 to 15. As a rule they are less extensive, and cause minor disturbances or go unnoticed. In later life rotation of loops of bowel within the hernia may produce a relatively acute obstruction. About half these hernias have sacs. Migration of abdominal contents is not extensive. Sometimes a portion of transverse colon, less often loops of small intestine or portions of omentum become incarcerated. In infancy and childhood the liver may be found in the sac.

This condition sometimes is noticed in young children by the demonstration of a bowel loop in the retrosternal area. Later the herniation can be identified by the presence of masses in the cardiophrenic angles, more often on the right side. These may move with respiration. If intestinal contents are present, the diagnosis is readily made. However, when only omentum is in the sac diagnostic pneumoperitoneum may be necessary. Occasionally the transverse colon is elevated in its midportion, producing a "batwing" appearance. Morgagni hernias often are found incidentally during chest or gastrointestinal examinations. In 12 patients seen by me, only 2 had symptoms sufficient to call the referring physician's attention to this possibility. However, incarceration, obstruction or strangulation of intestinal loops has been reported in from 10 to 15 per cent of these patients.

Considerable variation exists in the appearance of the paracardiac and retrosternal shadows. These sometimes are quite small, simulating epipericardial fat pads except for their lateral convexity. The shadow may be of uniform density, but with the entrance of intestinal loops, gas and fluid shadows appear. Occasionally little or no protrusion is seen to either side of the heart. Overpenetrated frontal and lateral films disclose the point of origin of the hernias quite often in adult patients, less so in children. Infrequently double hernias are found. Such a case (Fig. 7) was a 64-year-old man who complained of recurrent cramps. Bowel sounds were heard over his left hemithorax. Chest roentgenograms disclosed a bowel-like shadow in the right paracardiac area. Gastrointestinal examination revealed the transverse colon lifted into the foramens of Morgagni on either side of the midline. A portion of stomach protruded into the left retrosternal area. The diagnosis was confirmed at operation.

Incomplete fusion of various segments of the diaphragm form the basis for rather frequent hernias on either side. Such defects range from minor ones to complete absence of the diaphragm, which is represented by only a remnant of musculotendinous tissue at the periphery of the chest. We observed 29 such cases, 18 on the left and 11 on the right side, of such variance in size that the

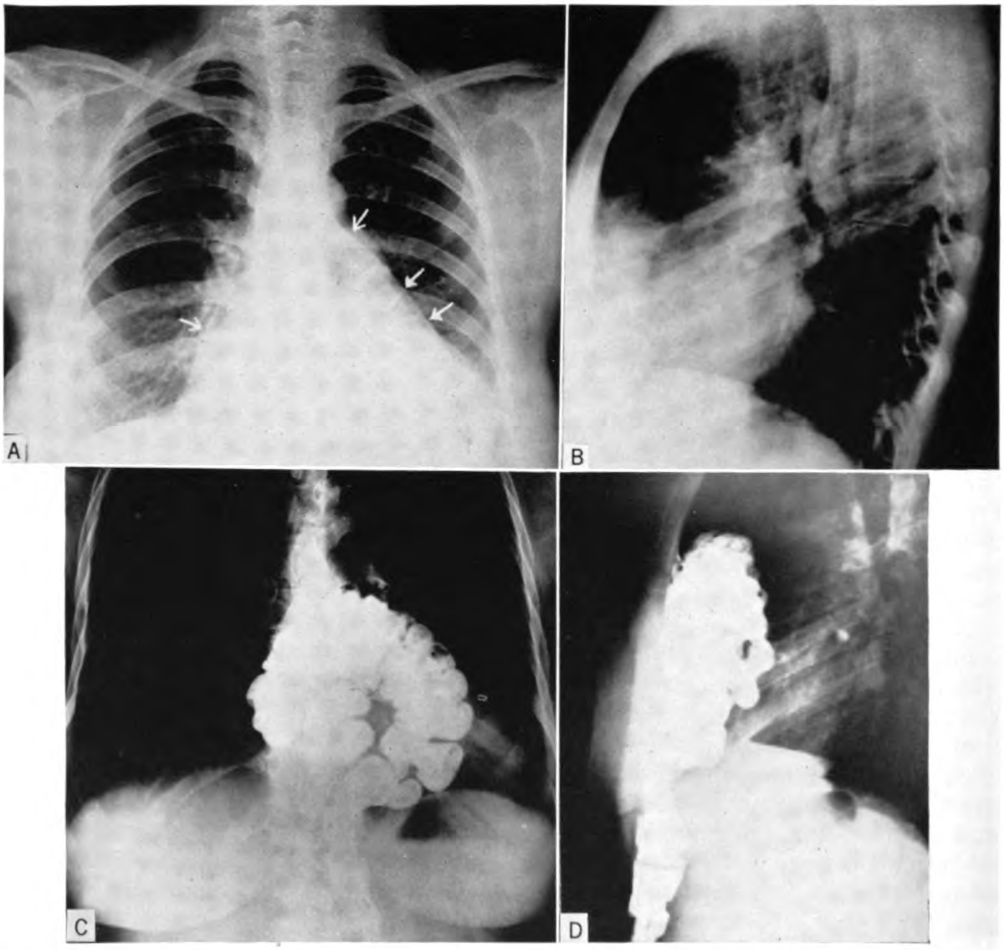

FIG. 6.—*A*, Hernia through left foramen of Morgagni. Teleroentgenogram of a 30-year-old woman with no chest or abdominal complaints. Film made during investigation for postpartum fever. A gas-containing loop of bowel with haustral indentations is seen overlying the heart (arrows).

B, Right lateral view, same patient. The gas-containing bowel is in the anterior aspect of the chest.

C and *D*, Erect anteroposterior and lateral chest roentgenograms 10 hours after a barium meal. The colon is in a large left foramen of Morgagni hernia.

defects could be referred to only as large, medium and small.

The time of onset of symptoms depends largely upon the displacement of abdominal contents into the thorax. The condition often is discovered in the first 2 days of life with dramatic symptoms of cyanosis, dyspnea and inability to feed. Vomiting and symptoms of intestinal obstruction are noted as well. About half the cases seen fell into this category, the others being diagnosed later in childhood and in adult life. A wide variety of visceral displacements are possible. On the left side the lesser and large bowel frequently enter the pleural cavity, and less often stomach, spleen, kidney and once in a while pancreas also are found. Rarely the left lobe of the liver finds its way into the left chest. Incarceration of loops of bowel with rotation may be followed by symptoms of obstruction and strangulation.

Defects seen in adult life always raise the question as to whether they are congenital or traumatic in origin. These are diagnostic

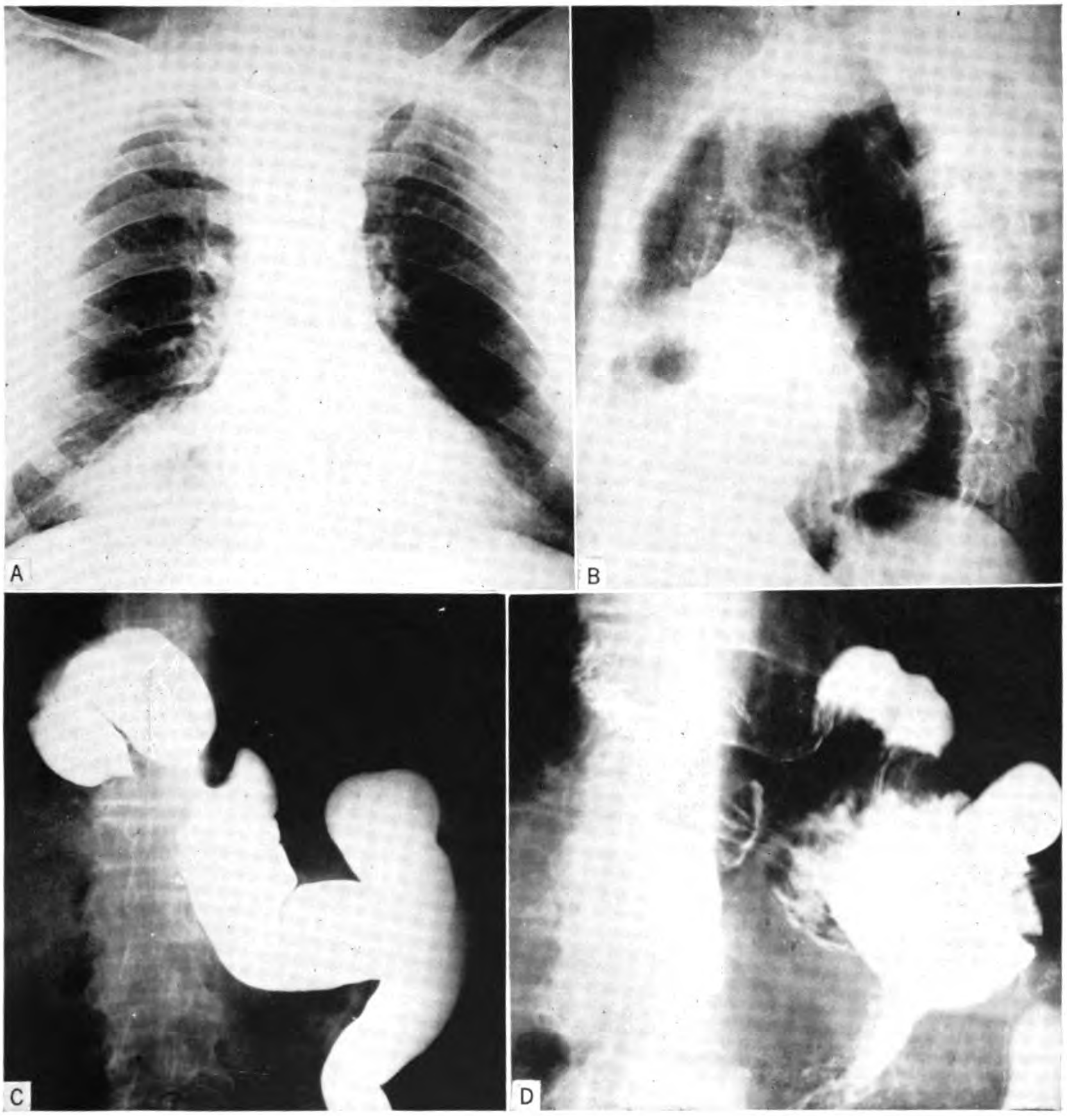

FIG. 7.—*A*, Double Morgagni hernia. Teleroentgenogram of a 64-year-old man with crampy abdominal pain. Bowel sounds audible over left chest. A gas-containing structure is present in the right lower lung field adjacent to the heart.

B, Right lateral chest film. The gas containing structure is anterior to the heart.

C, Barium enema examination reveals transverse colon within the thorax. Note the constriction of the colon as it enters the hernia and the pointed appearance of the bowel proximally. We were unable to get the barium into the proximal transverse or the ascending colon.

D, During a gastrointestinal examination made while barium was still in the bowel from the previous barium enema, transverse colon is seen in the lower chest both to the right and left of the spine in a double Morgagni hernia. Verified at operation.

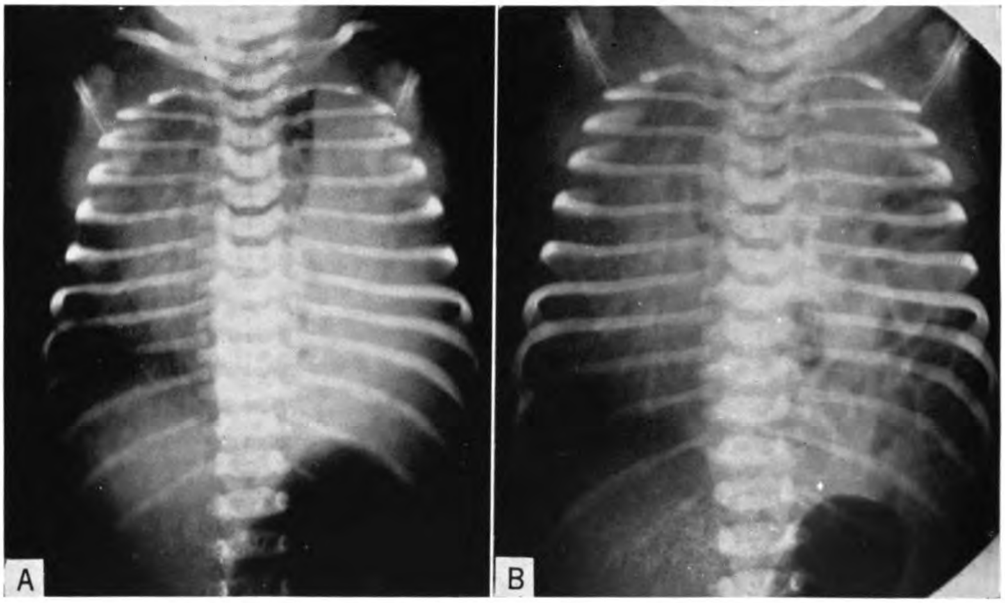

FIG. 8.—*A*, Left diaphragmatic hernia in a ½ hour old infant with respiratory distress. The heart and mediastinum are displaced to the right. Air is present in the stomach, but not in the bowel.

B, Same patient 2 hours later. Gas in the small intestine visualizes these bowel loops in the left chest. At operation a large defect was present in the dome of the diaphragm.

problems, often complicated by bizarre patterns of visceral migration and intestinal obstruction. Cases such as these were mentioned by Poppel, who reported a 62-year-old woman with herniation of the stomach, jejunum, duodenum, colon and pancreas within a sac. Rollandi found 2 cases of herniation of the spleen through the diaphragm. Campbell reported a 79-year-old man with intestinal obstruction predicated upon congenital absence of the left diaphragm, the initial diagnosis in this patient being pneumothorax. In the older age group gastrointestinal bleeding may produce the initial symptoms. The pain of incarcerated hernia may be severe enough to simulate coronary artery disease, and severe respiratory embarrassment also may be produced.

An interesting anomaly noted with congenital diaphragmatic defects is aplasia or absence of the ipsilateral lung. This condition is suspected when symptoms appear soon after birth, and when the herniation is extensive. Occasionally both lungs are maldeveloped.

Herniations through the right diaphragm are less frequent. As with left diaphragmatic hernias, the extent of the malformation varies considerably. Passage of abdominal contents into the right hemithorax may be extensive. The entire liver may enter, or only a portion bulges upwards, the remaining diaphragm acting as a restraining rim. In such cases the liver simulates a mass with a superior sharply demarcated margin, or a mediastinal tumor.

Not infrequently other congenital defects are found, particularly changes in the length and disposition of the peritoneal reflections. Concomitant cardiac or urinary tract malformations also appear.

The diagnosis of an extensive diaphragmatic hernia often is made soon after birth on conventional chest roentgenograms. When respiration has occurred air is seen in the stomach and bowel. In 2 cases with postmortem examinations gas was observed in the stomach, which was in the midline. Both lungs were densely opaque. Necropsy disclosed complete atelectasis of both lungs and congenital absence of the left diaphragm. The stomach was inverted and lay in the

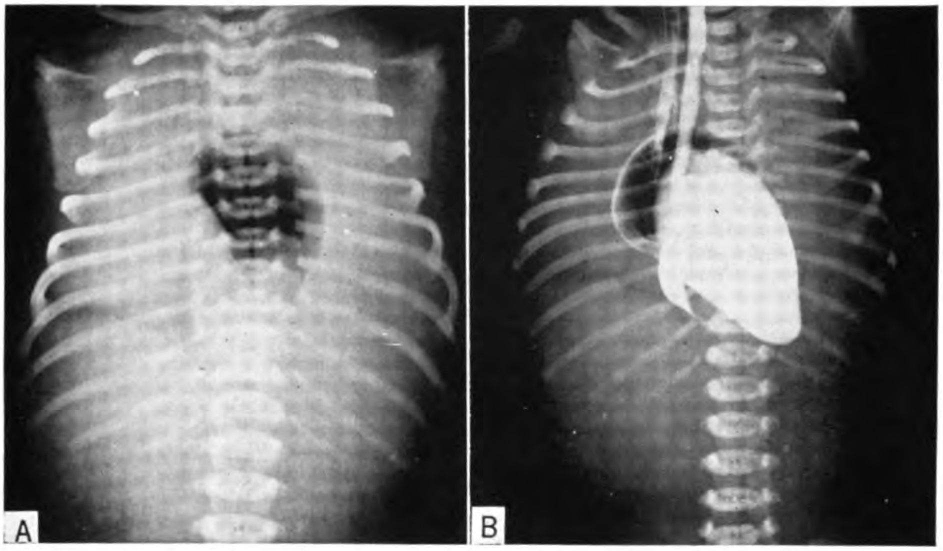

FIG. 9.—*A*, Left diaphragmatic hernia in a newborn who lived for about ½ hour. An intrathoracic stomach is present. The lung fields are dense.

B, Same patient soon after death. A nasogastric tube was introduced and barium injected. The esophagogastric junction is below the diaphragm. Some barium is present in the trachea. At necropsy the stomach, small bowel, spleen and left lobe of the liver were in the left hemithorax, entering through a defect in the left diaphragm. The left lung was hypoplastic, the right completely atelectatic.

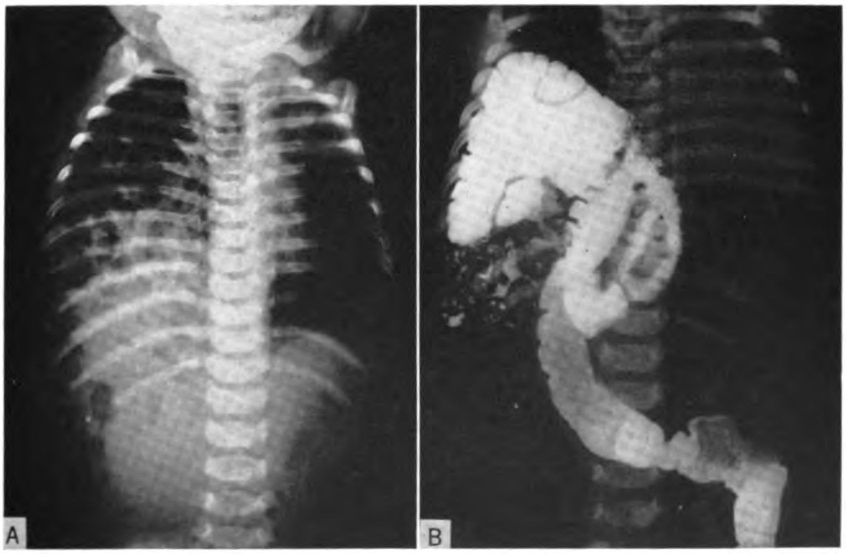

FIG. 10.—*A*, Right diaphragmatic hernia in an 8-day-old infant. The right hemithorax contains numerous gas-filled loops of small and large intestine.

10*B*, Same patient, 5 hours after a barium meal. At operation a defect in the middle of the right diaphragm extending posteriorly was found and repaired.

mediastinum. The lesser bowel was in the left chest. The liver had rotated, and together with the gallbladder was in the left chest. Similar changes were present in both infants, which had lived for about half an hour.

Occasionally symptoms are not apparent until the child is several months old. First attention may be directed to the condition when auscultation reveals bowel sounds over the chest. Such patients may be quite comfortable, or may complain of intermittent pain.

Infrequently a communication is present between a diaphragmatic hernia and the pericardial sac. One of our patients with congenital absence of the right diaphragm had a loop of bowel enter the pericardium. This became strangulated and at operation and subsequent necropsy both large and lesser bowel were present. This patient also had a hypoplastic right lung and atelectasis of the left lung. Wolfson and Crawshaw have reported on the pericardial components of diaphragmatic hernias in detail.

Following repair of a diaphragmatic hernia it is advisable to obtain chest roentgenograms to exclude the appearance of pneumothorax. In our group there were 2 infants who developed respiratory distress after operation which was found to be due to pneumothorax. This could not be controlled, and both youngsters expired.

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HIATAL HERNIAS

Esophageal hiatal hernias occur at any age, but are far more frequent in elderly individuals. The reported incidence varies

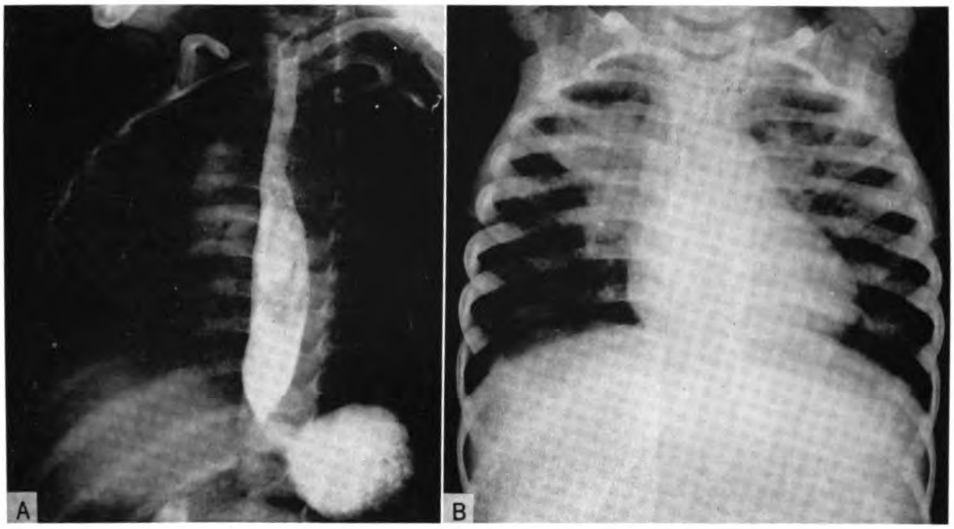

FIG. 11.—*A*, Chalasia with rapid and profuse reflux of barium into the esophagus in a 6-month-old infant. *B*, Same patient 3 days later, with diffuse bilateral aspiration bronchopneumonia.

from 1 to 15 per cent, depending upon the source of the data. Necropsy series have a lower occurrence rate inasmuch as sliding hiatal hernias as a rule are not diagnosed then. In common with the congenital hiatal hernias of infancy, adult lesions originate in a congenital weakness of the esophageal hiatus. Normally this passageway ranges in size from an opening permitting the passage of 2 fingers to a much larger one, and is situated mainly in the right diaphragm. Today's opinion is that the muscular action of the diaphragm and the hiatal margins play no effective part in gastric continence.

Congenital shortening of the esophagus with partial intrathoracic stomach is included with hiatal hernias, even though some authorities consider this developmental disorder outside this type of defect. Partial intrathoracic stomach is seen in association with chalasia, when reflux of acid gastric contents into the esophagus results in peptic esophagitis and shortening of the tube. The principal cause of chalasia in infancy is attributed to temporary neuromuscular dysfunction of the diaphragm and of the intrinsic gastroesophageal musculature. The differentiation of true congenitally shortened esophagus and partial intrathoracic stomach from chalasia and peptic esophagitis often is difficult. Chalasia is more frequent, with a clinical picture of free regurgitation of gastric contents through a relaxed cardia, weak

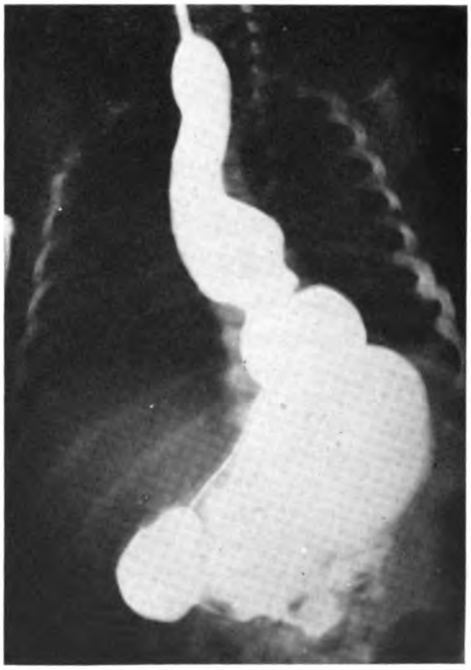

FIG. 12.—Chalasia of esophagus, with intrathoracic stomach in a 6-week-old infant who also had hypertrophic pyloric stenosis.

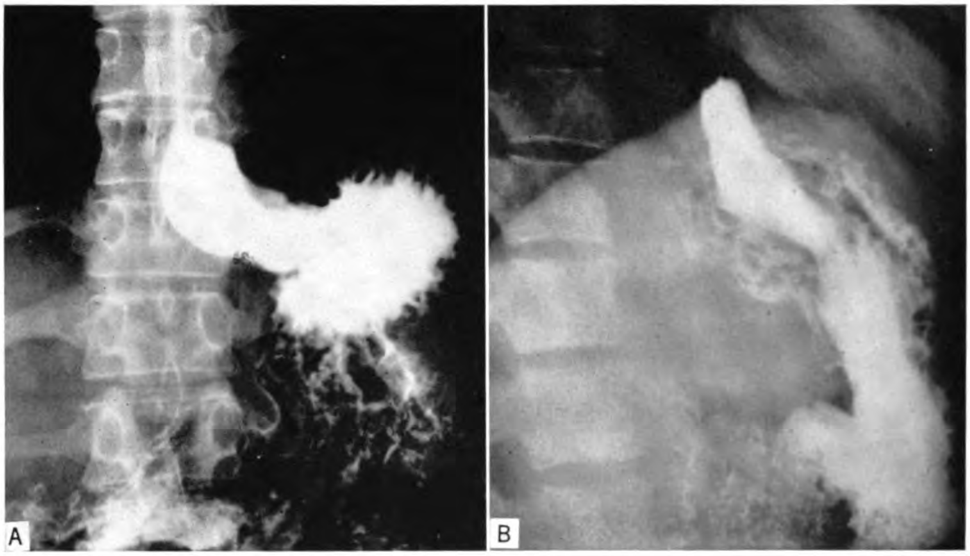

FIG. 13*A* and *B*.—Filling of cardiac antrum of stomach in supine and right anterior oblique Trendelenburg positions simulating sliding hiatal hernia.

or absent esophageal peristalsis, flaccidity of the esophagus and a pronounced tendency for regurgitation and vomiting when the patient is recumbent. To a large extent this is correctible by maintaining a semi-erect or posture after feeding.

Sliding hiatal hernias occur when an incompetent hiatus permits transitory upward displacement of the cardia with the patient recumbent. As a rule this is not apparent in the erect posture. The extent of sliding herniation is very variable. The position of the esophageal insertion is not necessarily constant with this deformity. In some the cardio-esophageal orifice remains below the diaphragm, and in others it passes through the hiatus to enter the thoracic cavity with the herniated cardia. If an incompetent cardio-esophageal mechanism permits reflux of gastric contents, peptic esophagitis may follow. Sliding hiatal hernias may or may not be contained in hernial sacs. There is more of a tendency for the smaller hernias to have sacs, and these are considered true hernias. More often no sac is present, constituting the so-called false hernias.

By far the most frequent form of hiatal hernia is the para-esophageal variety. These, too, are associated with congenital weakness of the hiatal orifice and its associated ligamentous structure. Loss of resiliency of fibrous and elastic tissues in later life, obesity, pregnancy, and thoracic deformities are all contributory factors to the marked prevalence of hiatal hernias in adults.

The relationship of the lower esophagus to the hiatal opening and the herniated stomach is variable, and is dependent upon the presence of peptic esophagitis as well as the extent of the hernia. In some patients a considerable portion of the stomach lies in the mediastinum, with the distal esophagus parallel to the medial and posterior portion of the stomach, below the level of the hiatus. In others, the esophagus may remain lateral to the stomach and be situated above the esophageal hiatus. With shortening of the esophagus incident to peptic esophagitis, the cardio-esophageal junction may be found at the apex of the herniated stomach. With larger hernias in which a major portion of the stomach finds its way into the thoracic cavity, the esophagus is often displaced posteriorly and its opening into the stomach is well below the rotated greater curvature, which assumes a superior position.

While hiatal hernias most often contain stomach alone, they also may be of the com-

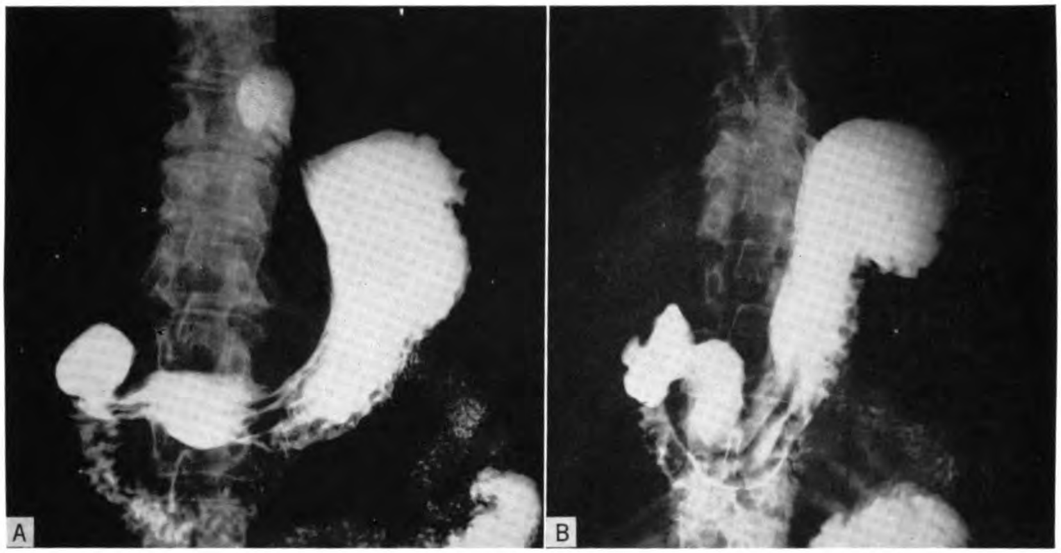

FIG. 14.—*A*, Small sliding asymptomatic hiatal hernia in a 65-year-old woman. This was not seen on erect films or fluoroscopically. Postero-anterior film.

B, Same patient in anteroposterior Trendelenburg position. The hernial sac did not fill on this view.

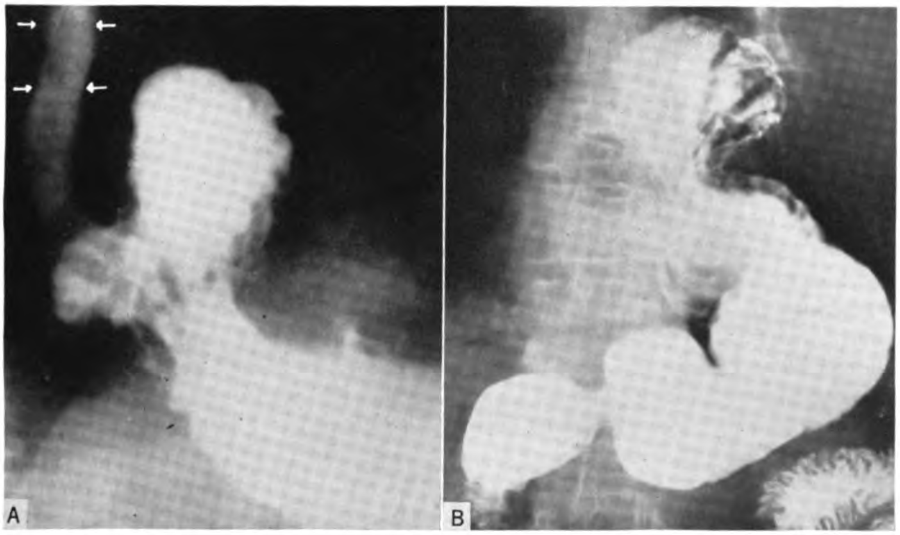

FIG. 15.—*A*, Paræsophageal hiatal hernia in a 55-year-old man, with the cardio-esophageal opening slightly above the level of the diaphragm, as seen on erect fluoroscopy and recorded on spot film.

B, Same patient, supine Trendelenburg roentgenogram shows a fixed hernia with no reflux into esophagus.

pound variety with colon, spleen, kidney and pancreas also present.

The symptoms of hiatal hernias in infancy usually are related to difficulty in respiration and feeding, with regurgitation, vomiting, choking and aspiration most prominent. Peptic esophagitis may be heralded by the refusal of the child to eat, by hematemesis or blood in the stool. Symptoms incident to pressure of the distended stomach on adjacent thoracic viscera are more common in later life, when biliary or coronary artery disease may be simulated. Gastrointestinal disturbances are relatively frequent, but no definite pattern can be elicited in the patient's complaints other than the occasional history of relief when erect and symptoms of heaviness and distress when recumbent soon after eating. A sharp pain in the back is an indication of esophagitis. Another fairly common complaint is weakness, accompanied by a hypochromic macrocytic anemia. Bleeding from peptic esophagitis or from ulcerations within incarcerated stomach is not uncommon.

A diagnosis of hiatal hernia occasionally is made on routine chest roentgenograms by identification of a retrocardiac radiolucency in which fluid levels may be present. Occasionally, a hiatal sac is large enough to overlap the heart borders. A double fluid level with a hernia should alert one to the possibility of torsion of the stomach within the sac, a precursor of incarceration, volvulus and strangulation.

Fluoroscopic observation of the first swallow of barium is useful in identifying the cardio-esophageal orifice, and should be done first with the patient erect. Various pressure maneuvers such as bending sharply to the side, elevating one leg or straining have been advocated as helpful in eliciting the presence of a hernia. Indeed, it is quite frequent to observe a perfectly normal appearing esophagus in erect studies followed by a definite sliding hernia with the patient recumbent. Films made with the patient in the Trendelenberg position have not increased the incidence of positive examinations, and are not necessary in many cases. The position which seems to bring out the greatest number of sliding hernias is the right prone oblique position, in which the barium flows towards the cardio-esophageal opening as the most dependent portion of the stomach. The rugæ of the lower esophagus and stomach should be carefully checked for changes of esophagitis, principally narrowing the ulceration. One must guard against misinterpreting a phrenic ampulla as a hiatal hernia, particularly when studies are made with the patient erect in forced inspiration. Under such circumstances a phrenic ampulla often persists long enough for a well-formed fluid level to simulate that seen in a hiatal hernia. The entrance of barium into the cardiac antrum of the lower esophagus also simulates a sliding hernia. This can be differentiated in many instances by the fact that the distended lowermost portion of the esophagus remains below the level of the diaphragm.

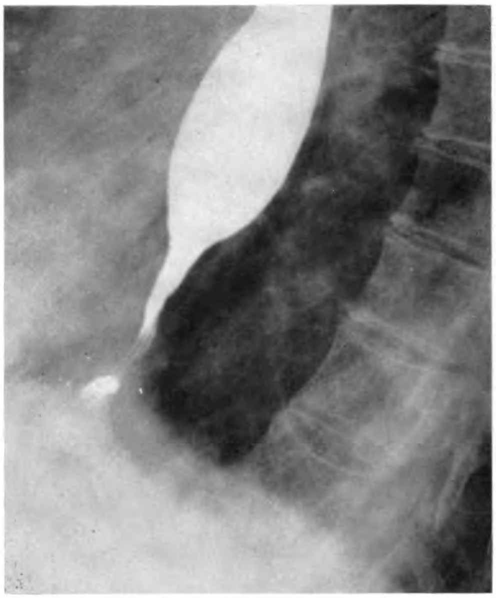
FIG. 16.—Peptic esophagitis in a 58-year-old patient with dysphagia and weight loss. The distal esophagus is tapered and narrowed. At esophagoscopy leucoplakia was visible. The patient was known to have had a hiatal hernia for about 4 years.

An infrequent but dramatic complication of hiatal hernia is gastroduodenal obstruction. The stomach, when rotated within the sac, becomes markedly dilated, and gangrene and perforation follow vascular impairment. This is more common in elderly patients. Strangulated hernias are more frequent in the post-traumatic types of diaphragmatic impairment. Carter and Giuseffi, in a review of 43 cases of strangulated diaphragmatic hernia, found 85 per cent to be of traumatic origin. Another infrequent complication is mild dysphagia due to spontaneous invagination and disinvagination of the esophagus into the herniated stomach.

Bleeding with hiatus hernia follows ulceration of mucous membrane of the incarcerated stomach, or peptic esophagitis. Occasionally constriction around the hernial ring causes gastritis and venous congestion. Trauma to

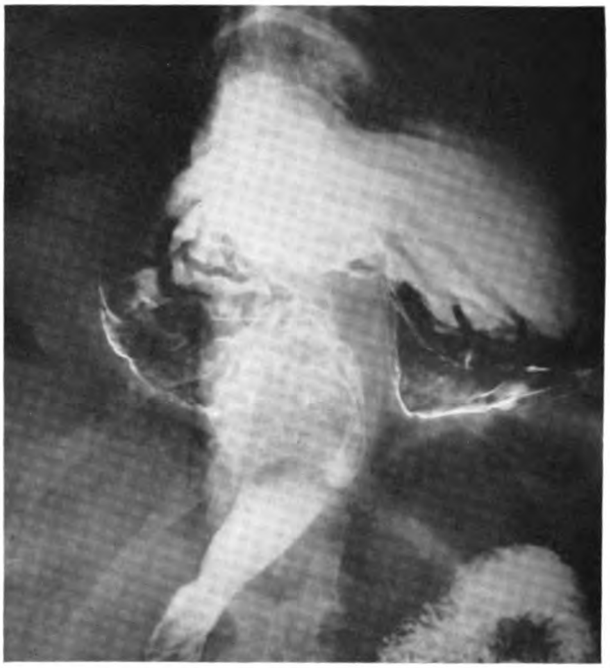

FIG. 17.—Gastric volvulus in a fixed hiatal hernia in a 65-year-old woman. This was known to have been present for at least 8 years and was asymptomatic.

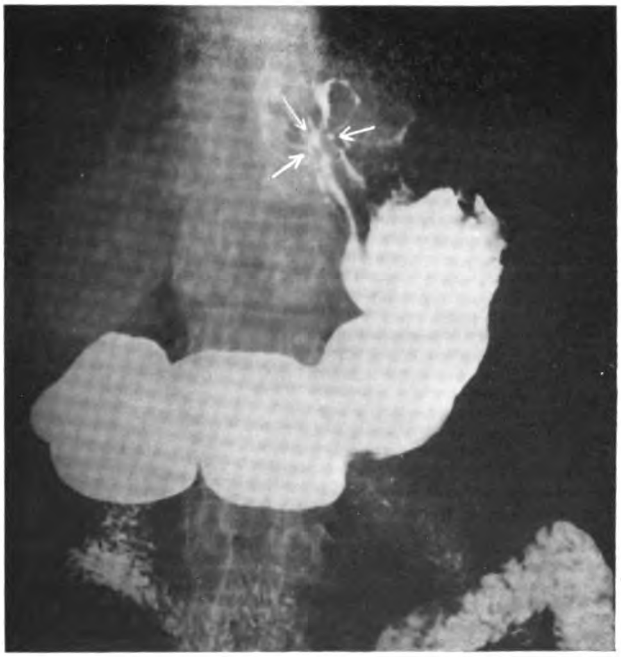

FIG. 18.—Ulcer in a hiatal hernia in a 54-year-old woman with gastric bleeding and upper abdominal pain. Note the convergence of gastric folds towards the ulcer crater (arrows).

the swollen, congested mucosa may cause bleeding, or ulcers may form along this congested ring. These occur somewhat more frequently along the lesser curvature of the stomach as it passes through the esophageal hiatus.

The association of neoplastic disease with hiatal hernia has been observed relatively often. Smith collected 96 such cases from the literature, and added 12 more. The radiologic demonstration of this change sometimes is quite difficult. In 2 cases seen by us the lesion was suggested by a small filling defect in the herniated stomach along its greater curvature, and in a third case only a rigidity and shortening of the involved wall led to the diagnosis.

This subject should not be dismissed without reiterating Harrington's precautions in evaluating the anatomy of hiatal hernias. There are decided limitations in the radiologic evaluation, and often it is necessary to obtain esophagoscopic and gastroscopic assistance. Not infrequently the true nature of the lesion is disclosed only at surgery, which should be reserved only for those with symptoms warranting intervention.

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EVENTRATION OF THE DIAPHRAGM

By definition, eventration indicates protrusion of abdominal contents outside the abdomen. Diaphragmatic eventration, which may be partial or complete, designates a congenital muscular maldevelopment with upward bulging of the involved side. Partial eventration usually involves the central medial aspect of the right diaphragm, but also occurs infrequently on the left side. The upward bulging permits the underlying viscera to protrude well into the thorax. As the liver molds itself into this abnormal space an accessory lobe evolves. While this appears to be in the thoracic cavity, it nevertheless is subdiaphragmatic in position.

Eventration of the diaphragm appears in all age groups. Among the terms used interchangeably to describe this condition are partial eventration, circumscribed relaxation of the diaphragm, loculation of the diaphragm, scalloped diaphragm and congenital hernia of the diaphragm. Other terms employed include superior accessory or anomalous lobe of the liver and diaphragmatic herniation of the liver.

Diaphragmatic eventration sometimes is identified in the newborn. Of 8 such cases in my experience, 3 came to necropsy within a few days after birth. These had practically complete eventration of both diaphragms, with the lobulated relaxed leaves high in the thorax bilaterally. Postmortem examination showed that the diaphragm, while complete, was reduced to a thin, fibrous sheet without muscle. Concomitant congenital malformations of the heart, the genitourinary and gastrointestinal tracts also were present. In 2 cases fetal atelectasis of both lungs was quite pronounced. In the other cases no symptoms could be attributed to the eventration.

Partial eventration of the right diaphragm sometimes simulates a mediastinal mass. When confined to the medial aspect of the diaphragm there may be difficulty in distinguishing it from a Morgagni hernia. Nayer reported a case of right-sided stomach associated with eventration of the diaphragm which simulated hydropneumothorax.

In adults partial eventration of the right diaphragm with corresponding hepatic deformities is regarded mainly as an incidental and rather frequent observation. However, Vogl and Small recorded 3 cases in which herniated portions of liver became strangulated with symptoms of intermittent incarceration.

Complete eventration of the diaphragm is more frequent on the left side, and as a rule is relatively symptomless. The motion may be studied fluoroscopically, but better evaluation of this condition can be made by means of roentgenkymography. These studies show that the alteration of the movement of the involved diaphragm is not exactly parodoxic in the sense that it is precisely opposite in phase to the motion of the normal side. Rather, the movement is impaired and widened, reflecting the impairment of the

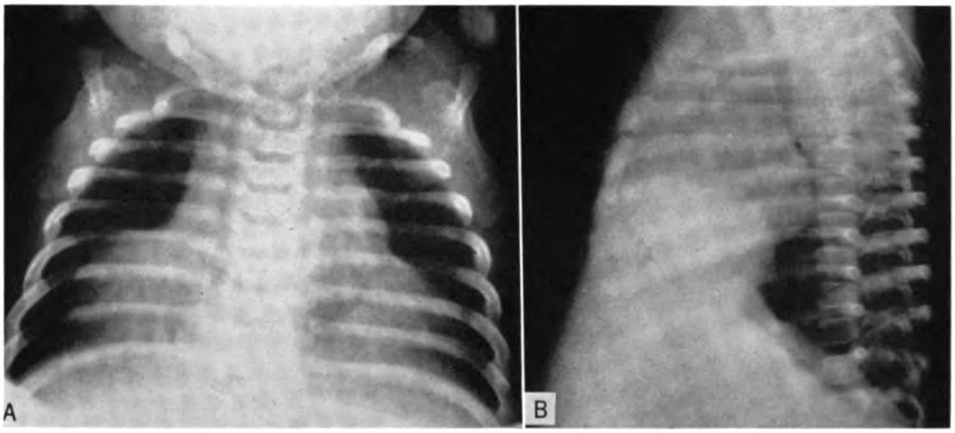

FIG. 19*A* and *B*.—Eventration of the right diaphragm in a 6-week-old infant, diagnosed preoperatively as a pulmonary or mediastinal tumor. The liver was elevated and presented a dome-shaped deformity corresponding to the weakened anterior diaphragm.

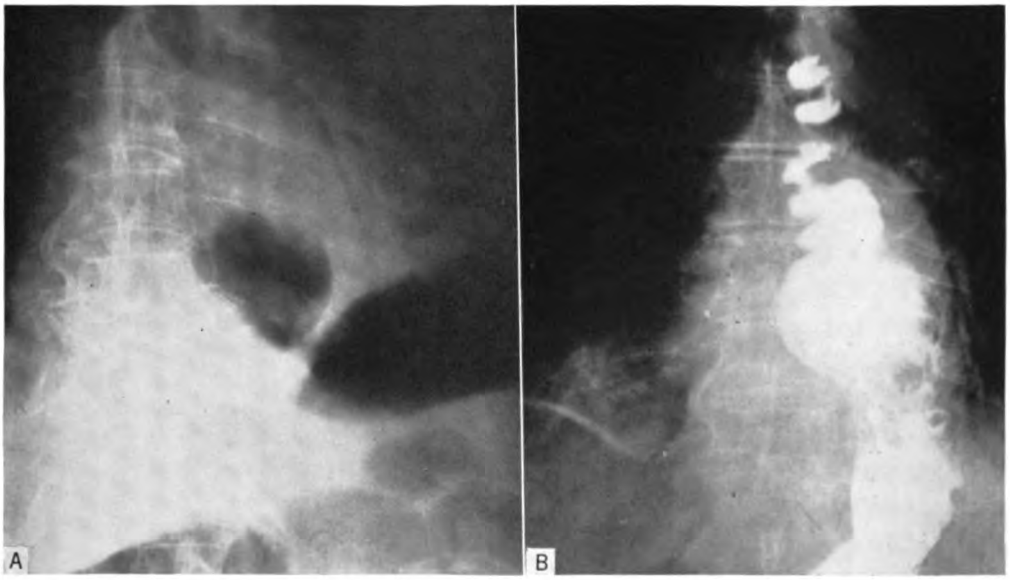

FIG. 20.—*A*, Partial eventration of medial aspect of the left diaphragm diagnosed as a hiatal hernia. Scout film shows gas in elevated stomach. Patient was an 80-year-old male with jaundice due to impacted common duct stone.

B, Same patient with barium in esophagus. Tertiary contractions of the esophagus are present. At necropsy the medial portion of the left diaphragm was thinned out into a delicate sheet with no muscle. The stomach and spleen were pushed up into this sac alongside the heart. No defect was present in the diaphragm.

muscle. In evaluating diaphragmatic muscular abnormalities, one must remember that previous phrenic nerve operations will influence the position and range of motion of the involved leaf. In infancy, birth trauma resulting in Erb's palsy also can be associated with phrenic nerve injury and concomitant diaphragmatic alterations. No association with acute abdominal disorders appears in these patients.

Partial eventration of the diaphragm in adults occasionally simulates hiatus hernia.

Gastrointestinal tract examination reveals the stomach to be elevated and often rotated upon itself, simulating organo-axial volvulus. The duodenum may be elevated and drawn to the left of the midline, thereby elongating the loop. The duodenal bulb is then pointed downwards. Compressive changes in the adjacent lung are sometimes produced by eventration, with shift of the heart and mediastinum to the contralateral side. As a rule, this is asymptomatic, but in a few cases compression atelectasis of the base of the lung was observed.

Diffuse scalloping is not necessarily of clinical importance, and is not associated with abdominal symptoms. As with the single scallop, confusion in diagnosis may be encountered. In these patients a diagnostic pneumoperitoneum is important in identifying the inferior aspect of the diaphragm.

A particularly interesting case occurred in an 88-year-old man (Fig. 20) in whom a diagnosis of hiatus hernia had been made because his stomach was in the retrocardiac portion of the left chest. The lateral aspect of the left diaphragm was normal. At necropsy the inner aspect of the left diaphragm was reduced to a thin fibrous sheet. The cardia of the stomach projected into this sac, and behind it was the spleen. There were no symptoms referable to this disorder.

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CONGENITAL DEFECTS OF THE ABDOMINAL WALLS

Congenital herniations through the muscular walls of the abdomen follow defects of segmental development in which the muscles appear as thinned or partly absent structures. Umbilical hernias are common examples, the peritoneal sacs containing omentum or intestine bulging under the intact skin. Radiographic examination of such hernias is indicated occasionally when intestinal incarceration is suspected.

Inguinal hernias are frequent in infants. Acute intestinal obstruction often complicates this condition. Abdominal roentgenograms help establish the diagnosis by the characteristic pattern of gaseous distention and identification of a trapped loop of bowel in the hernia.

Major congenital umbilical defects are infrequent. Maldevelopment of the omphalomesenteric duct produces defects reflecting the site of persistence, so that when the entire duct remains open an omphalocele results. This is apparent at birth as a peritoneal sac uncovered by skin, muscle or fascia into which the abdominal contents prolapse. Smaller omphaloceles may be seen first when the cord structures slough away, revealing a small red mass at the umbilicus. Fecal drainage in the umbilicus is a characteristic sign, and the diagnosis is readily made after injection of radiopaque material reveals communication with the intestinal tract. Kittle, Jenkins and Dragstedt noted but 2 cases of omphalocele among 30,000 births.

Failure of obliteration of the proximal portion of the omphalomesenteric duct results in Meckel's diverticulum. This will be discussed separately.

Vitelline cysts appear when the intermediate segment persists. These cysts are attached to the ileum or the umbilicus or both by a fibrous thread.

A patent urachus is suggested when urine escapes from a small opening in the anterior abdominal wall. Depending upon the pat-

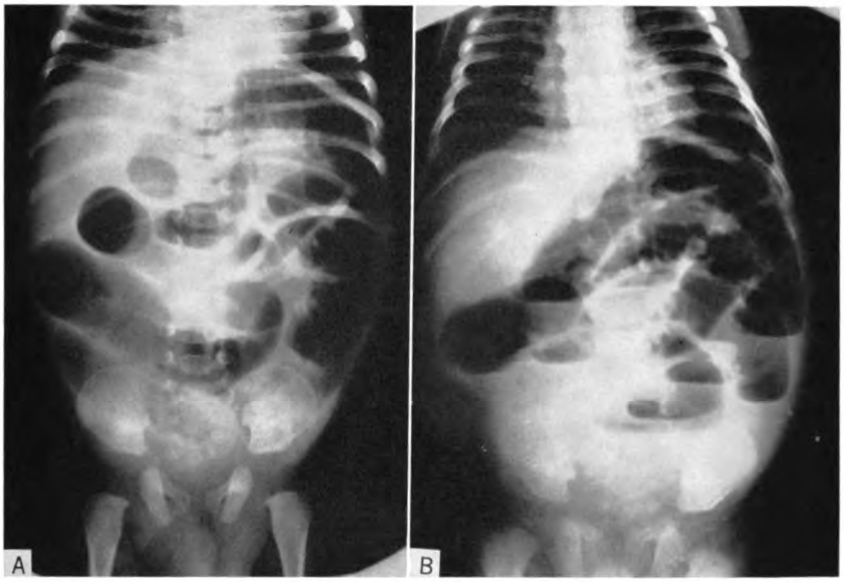

FIG. 21*A* and *B*.—Intestinal obstruction in a 7-week-old male infant with a right inguinal hernia. Supine and erect films reveal gaseous distention with multiple fluid levels. No gas is seen in the inguinal region in this patient, but a swelling was present on physical examination.

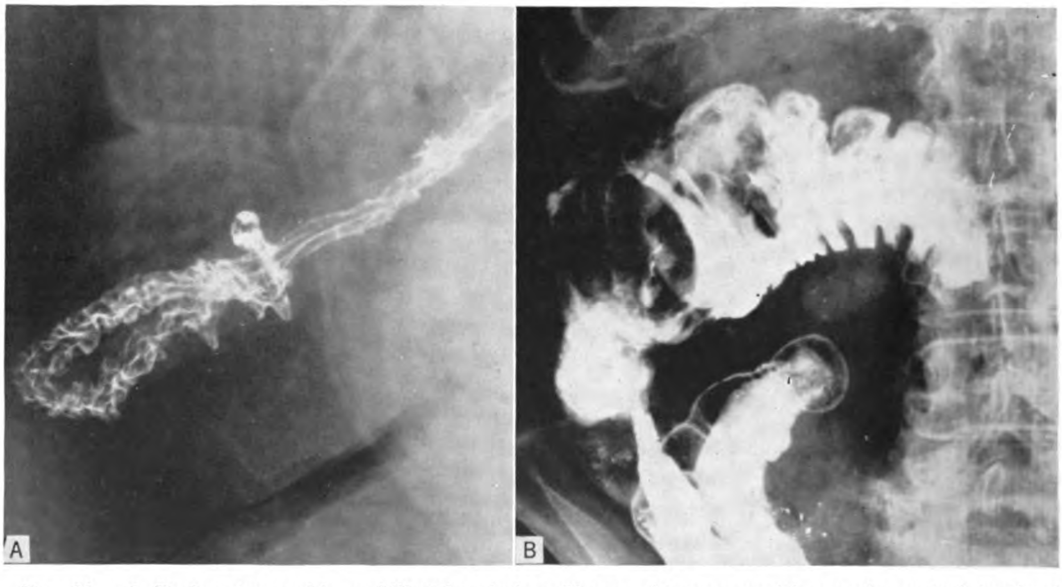

FIG. 22.—*A*, Barium trapped in umbilical hernia in a 72-year-old woman with anemia, gastrointestinal bleeding and weight loss. Post-evacuation film after barium enema, which showed obstruction at this site. *B*, Same patient, 5 hours after a barium meal. There is a large defect in the ascending colon. At operation a carcinoma was found at this site.

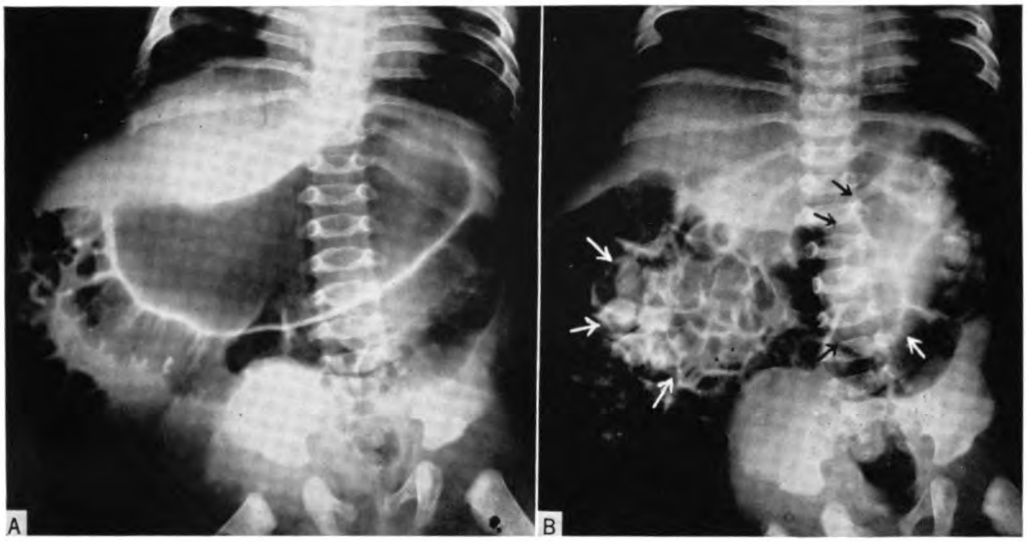

FIG. 23.—*A*, Scout abdominal film of a 6-month-old male infant with congenital absence of the right anterior abdominal wall. The distended stomach and colon are displaced towards the right side.

B, Same patient, 100-minute film, intravenous urography. The lesser bowel is distended by swallowed air and fills the left side of the abdomen. An enormous hydronephrosis is present bilaterally (arrows). The right kidney is displaced laterally.

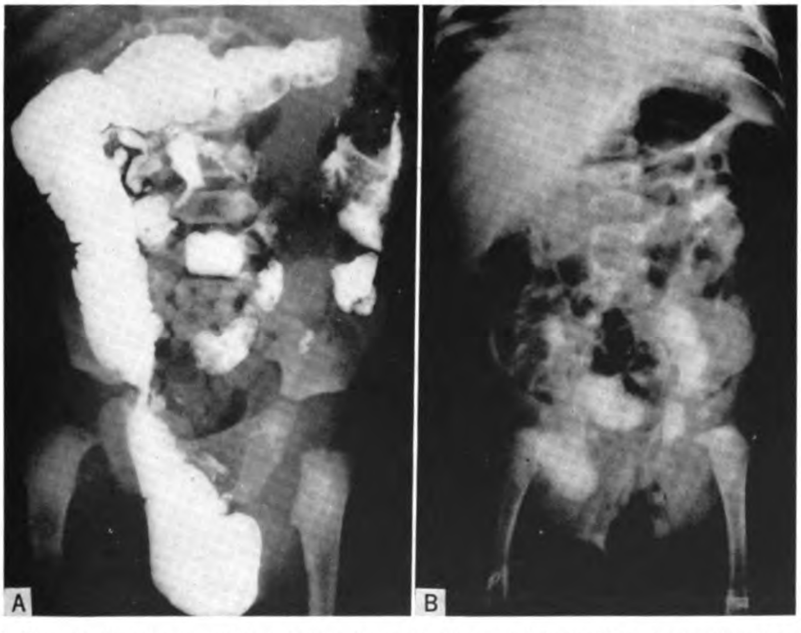

FIG. 24.—*A*, Scrotal hernia containing ileum in a 6-month-old boy. No obstructive symptoms were present at the time this film was made.

B.—Bilateral inguinal hernia containing bladder as well as small intestine on the right side in a 3-month-old boy. The bladder enters from the right side. This was not identified clinically.

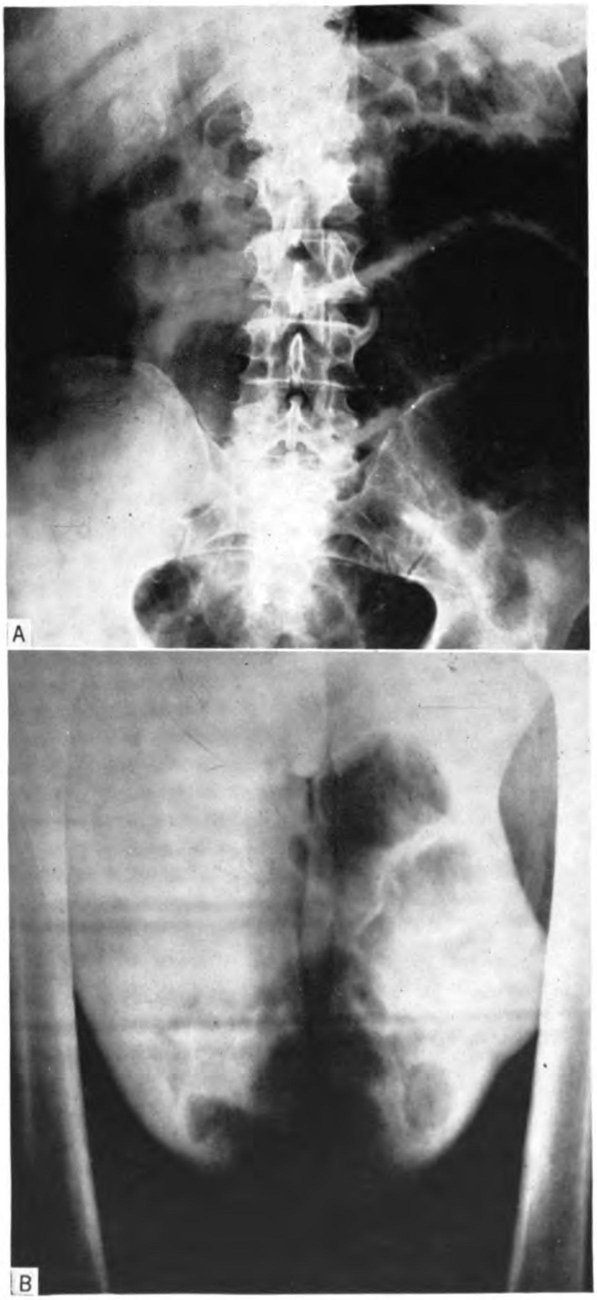

Fig. 25.—*A*, Partial small bowel obstruction in a 64-year-old man with an enormous scrotal hernia. Note the lesser bowel distention in the midabdomen to the left of the spine. The patient had been vomiting for 20 hours before this film was obtained.

B, Same patient, erect study showing scrotal hernia containing lesser bowel. The patient responded to small bowel decompression and operation was not required.

tern of urachal obliteration vari-sized cysts develop. Contrast material injection into these tracts will disclose either a cyst or bladder communication. The differential diagnosis between a draining urachus and patent omphalomesenteric duct is made often on clinical evidence, but injection of draining points with radioopaque material offers a simple radiographic method of accurate diagnosis.

Partial or complete deficiency of the anterior abdominal muscles is another relatively infrequent congenital malformation. This occurs chiefly in males, frequently together with urinary tract anomalies such as undescended testes, hydronephrosis, polycystic kidneys, ureteral and urethral deformities, hypertrophy and dilatation of the bladder and diverticula. Other congenital malformations present include club feet, pigeon breast, and various anomalies of the gastrointestinal tract.

The muscular changes vary from a marked thinning of the muscle with persistence of layers to complete absence of these structures. Often only the origins and insertions of the anterior abdominal wall muscles remain. The abdomen becomes relaxed, asymmetric and protuberant. The underlying viscera are readily palpated. Considerable gaseous distention is observed often. Intravenous urography is an essential part of the investigation of these children. In 2 of our 3 cases extensive urinary tract abnormalities were present. These included marked dilatation of the bladder, reflux from the bladder into the kidneys with pronounced hydroureteronephrosis, and displacement of the bladder into inguinal hernias.

The intrusion of small and large intestine into scrotal hernias is observed often clinically and radiologically in all age groups. In elderly patients this sometimes assumes massive proportions, with the hernias reaching the knees and containing practically all the intestine. Intestinal obstruction is a contraindication to the administration of barium to these individuals, either by mouth or by barium enema. If contrast examination is deemed essential a water-soluble preparation such as those used for angiocardiography should be used.

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MECKEL'S DIVERTICULUM

This sac represents a persistent proximal portion of the omphalomesenteric duct. It is a relatively common and usually insignificant anomaly. In 10,000 consecutive necropsies at the Johns Hopkins Hospital, Sloan and his co-workers found 103 Meckel's diverticula, all without symptoms. Nevertheless, Meckel's diverticulum can become important because of complications which appear as early as intrauterine life. Rosza and Gross reported a case of perforation of a Meckel's diverticulum *in utero*. Diagnosis was difficult because of abdominal distention. The presence of intra-abdominal mottled calcifications, moderate gaseous distension of the stomach and lesser bowel and the absence of gas from the colon suggested the radiologic diagnosis of meconium peritonitis from intestinal obstruction, presumably at the ileocecal junction. At necropsy 9 hours after birth a perforated diverticulum was found, with no free meconium in the peritoneal cavity although the peritoneal surfaces had the appearance associated with meconium ileus.

As a rule Meckel's diverticulum is a relatively wide-mouthed and short structure lined with intestinal epithelium. Heterotopic tissue often is present in the mucosa, predisposing to numerous complications. Gastric mucous membrane provides the basis for peptic ulceration with hemorrhage, perforation and peritonitis. Pancreatic rests likewise are relatively frequent. Malignant degeneration has been encountered occasionally. Intestinal obstruction predicated upon torsion about adhesions at the base of the diverticulum is a relatively frequent complication. Intussusception follows invagination of the diverticulum. These complications are not limited to children, and must be considered in the differential diagnosis of abdominal complaints of all age groups.

A radiologic diagnosis of Meckel's diverticulum is occasionally suggested by the presence of a relatively fixed, somewhat circular collection of gas in the lower mid-abdomen on scout roentgenograms. Recently the identification of calculi within the pouch with a characteristic folded, ribbon-like appearance has permitted a correct diagnosis. The diagnosis usually is suggested by the persistence of barium in the pouch on gastro-intestinal examination. Occasionally a Meckel's diverticulum will be identified by barium enema when barium enters the ileum and finds its way into the pouch. In rare instances injection of a sinus tract in the umbilicus leads to a Meckel's diverticulum at the junction of the omphalomesenteric duct and the ileum. There is doubt as to whether barium should be used in the presence of possible perforation or intussusception. The use of water soluble contrast material is suggested in these cases. The fact that Meckel's diverticula are so commonly found at necropsy and so infrequently observed radiologically indicates that this diagnosis is not made easily.

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GASTROINTESTINAL MALROTATIONS

Failure of normal rotation of the foregut in embryonal life results in incomplete descent of the stomach from its original thoracic situation. The stomach remains in the right thoracic cavity above the liver. Symptoms may appear early in life, or the condition may be entirely asymptomatic. Concomitant malformations of the diaphragm and gastrointestinal tract have been reported. The diagnosis is established by the abnormal situation of the stomach demonstrated either by the presence of gas or with barium.

Most rotational disorders involve the midgut, the bowel from the duodenum to the midportion of the transverse colon supplied by the superior mesenteric artery. From the third to the fifth week of fetal life the rapidly enlarging intestinal coils cannot be accommodated within the abdomen, and enter the umbilical cord. Later the midgut re-enters the abdomen. The prearterial segment which extends from the duodenum to the vitelline duct, returns first, passing from right to left under the superior mesenteric artery. The postarterial segment, from the vitelline duct to the midtransverse colon, passes from left to right in front of the superior mesenteric artery. Following this the cecum descends to the right lower quadrant. The visceral and parietal peritoneal leaves fuse to form a mesentery for the small intestine, attached obliquely to the posterior abdominal wall. Later the cecum and ascending colon are fixed in their usual positions in the right abdomen.

The defects with which we are now concerned occur in the tenth to eleventh week of fetal life. The impaired return of the intestines into the abdomen is associated with deficient mesenteric attachment along the posterior abdominal wall. The mesentery then is a rather long, narrow structure. The cecum rotates incompletely, remaining in the midline, or if partially rotated, comes to rest high in the right side of the abdomen under the liver. Normally the lesser bowel re-enters the abdomen and is situated so that the jejunal coils are in the left upper abdomen and the ileal coils descend to the right lower abdomen. Rotational abnormalities are present when the jejunum is situated in the right side of the abdomen, and the ileum is found in the left or middle position. An abnormally high or a movable cecum also indicates malrotation. Associated with disturbances of intestinal return are abnormal peritoneal bands, which represent distorted fusion or failure of absorption of abnormally placed peritoneal structures.

With a common mesentery the entire duodenum remains to the right of the midline instead of extending across the spine to the left side. The right colon remains in the left abdomen, with the cecum in the left iliac fossa. The jejunum generally lies on the right, and frequently part of ileum remains there as well. The terminal ileum then has to cross the midline to join the cecum on its right aspect. The position of the stomach, liver and pancreas is not changed. The colon sometimes appears to be normally placed, particularly when examined with the patient horizontal. Abnormal positions are seen better on erect studies in some case.

Not infrequently midgut malrotations are asymptomatic. To a great extent the clinical picture depends on the obstruction caused by abnormal mesenteric reflections impinging against the small intestine. Volvulus is possible when the mesenteric attachment to the posterior abdominal wall is deficient, permitting rotation of the bowel. This usually occurs in a clockwise direction and one to two complete turns may take place. The coexistence of compression and obstruction of the duodenum by mesenteric bands and volvulus is frequent. Identification of an abnormally situated cecum, usually high in the right abdomen, often is helpful in diagnosing malpositions of the small intestine and mesenteric anomalies.

Inversion of the duodenum is ascribed to persistence of the dorsal mesentery and a rotational error of the midgut which causes a mobile duodenum with either shortening

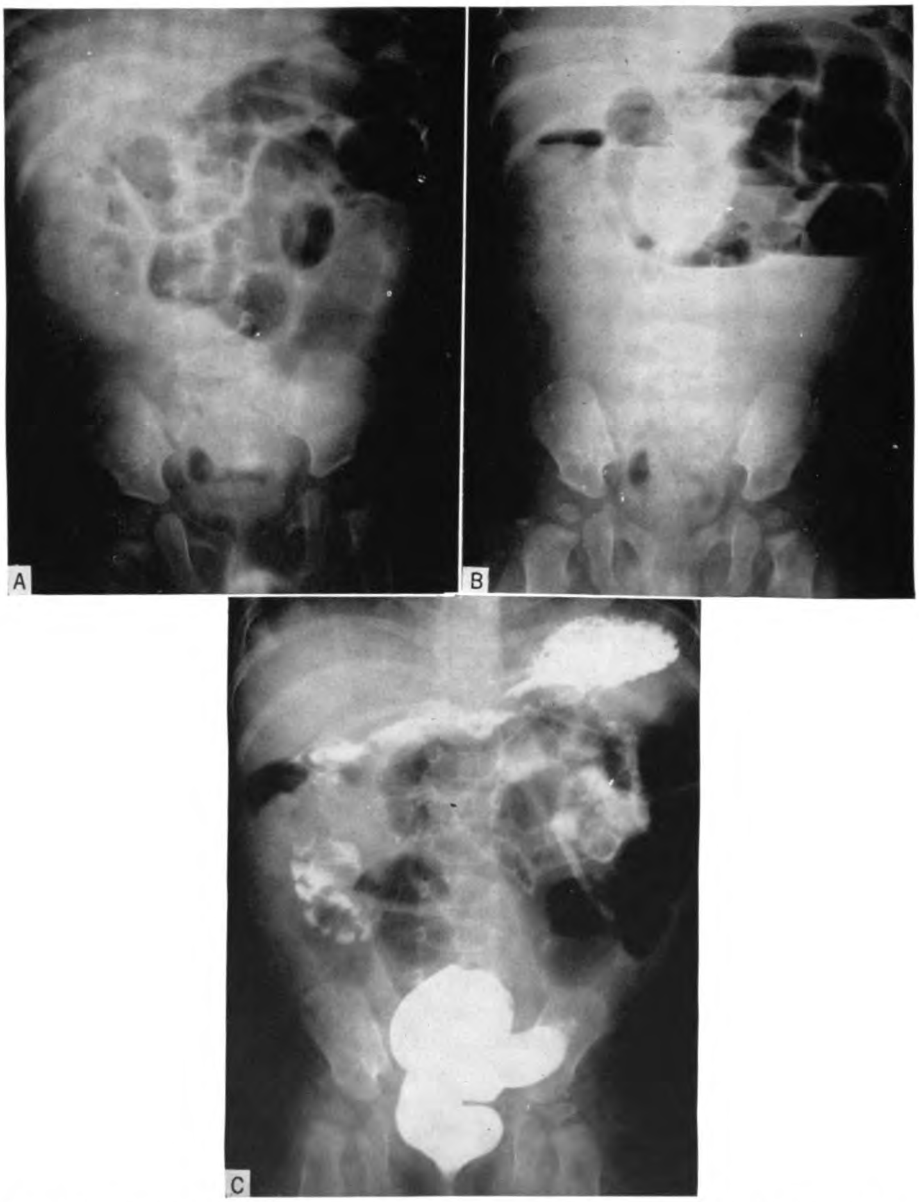

FIG. 26.—*A*, Jejunal volvulus in a 7-month-old female infant with vomiting. Supine film. Note the distended small intestine in the left upper and mid-abdomen.

B, Same patient, erect film. The distended lesser bowel loops are high in the left upper and mid-abdomen. No gas is present in the colon.

C, An enema with water soluble contrast medium (Hypaque) reveals no obstruction and the cecum is high on the right side. Thereupon 15 cc. of Hypaque was instilled into the stomach through a nasogastric tube. This shows slow passage through the distended upper jejunal coils. At operation a congenital band extended from the middle of the mesentery across the distal jejunum encircling the bowel, acting as a pivotal point about which the jejunum had twisted 180 degrees. Cecum high, on long mesentery, in the right upper quadrant.

or lengthening of the caudal duodenal segments. As a result the normal duodenal curve is reversed. The third portion, instead of turning to the left and crossing the midline, swings to the right. After taking an upwards course the sweep turns to the left and crosses the midline above the pancreas. This change usually is asymptomatic, but has been associated with nausea, discomfort, belching and epigastric pain. Jaundice and pancreatitis have been attributed to stasis in the duodenal loop.

Volvulus is the most dramatic complication of midgut malrotation. This can occur immediately after birth, often accompanied by duodenal obstruction. In early stages the midgut may be collapsed, but distension of the involved loops soon follows entrance of swallowed air or gas formation. Malrotations of the small intestine often are quiescent until adult life. Gross observed that most patients with complaints attributed to malrotation are seen in the neonatal period. Of his 156 patients, 86 were in the first month of life, 40 were from 1 to 12 months old and 29 were from 1 to 15 years old. Symptoms of obstruction were more acute in infants, requiring immediate operation. In older patients symptoms were more likely to be intermittent and recurrent.

Usually it is not possible to make a specific diagnosis of volvulus on radiologic evidence alone. Erect and supine scout films often reveal a nonspecific pattern of gaseous distension and fluid levels. Air insufflation of the stomach is helpful in identifying an obstruction in the duodenum or proximal jejunum. This can be better accomplished by the instillation of water-soluble contrast material into the stomach, quantities as little as 10 to 20 cc. being adequate to outline the stomach and lesser bowel. If volvulus is suspected, it is important to ascertain the location of the cecum either by the insufflation of air or water-soluble contrast material into the rectum.

Of the 15 cases of volvulus seen by us in infants up to 8 months old, 11 occurred before 1 month of age, the youngest being 6 hours old. This infant had acute symptoms of abdominal and respiratory distress with vomiting. Scout abdominal roentgenograms disclosed scattered distended lesser bowel loops and pneumoperitoneum (Fig. 27). Operation revealed a double rotation of the ileum with perforation of a gangrenous loop, and meconium peritonitis. In another 3-day-old infant with volvulus verified at operation pronounced intestinal distension was present. Within the distended bowel were granular shadows which at first glance suggested the possibility of meconium ileus. Another 8-day-old infant had bile stained vomitus which contained undigested food. This was attributed to an obstructive duodenal lesion even though scout films disclosed intestinal distension. The jejunal coils were identified in the right side of the abdomen. At operation the proximal ileum had rotated 360 degrees upon itself in a clockwise direction. In all 3 cases the cecum was high in the right upper quadrant.

In 2 cases concomitant congenital malformations were present. In 1, a 4-month-old infant, with jaundice and vomiting since birth there was congenital absence of the biliary tract. At operation the jejunum was found in the right upper abdomen and the cecum was under the liver. Another youngster had polycystic kidneys.

Intermittent symptoms associated with midgut malrotation is illustrated by a 14-week-old infant who had sporadic pain associated with distension and vomiting. Scout films showed no specific changes. Gastrointestinal examination revealed marked delay in the passage of barium through the duodenum, with only a small amount entering the jejunum. At operation the duodenal obstruction was seen to be caused by peritoneal bands which arose from the cecum, which was situated under the liver close to the midline. The bands extended from the cecum across the duodenum to the lateral right parietal peritoneum.

Our experiences with small bowel volvulus in adults has been predominantly with patients who had previous operations. In most cases there was no malrotation of the intestines nor cecal displacement.

Rotational defects of the colon beyond the midtransverse segment are infrequent. Occasionally elongation of the mesentery of the descending and sigmoid colon gives rise to dolichocolon, a redundancy sometimes associated with symptoms later in life. Malposi-

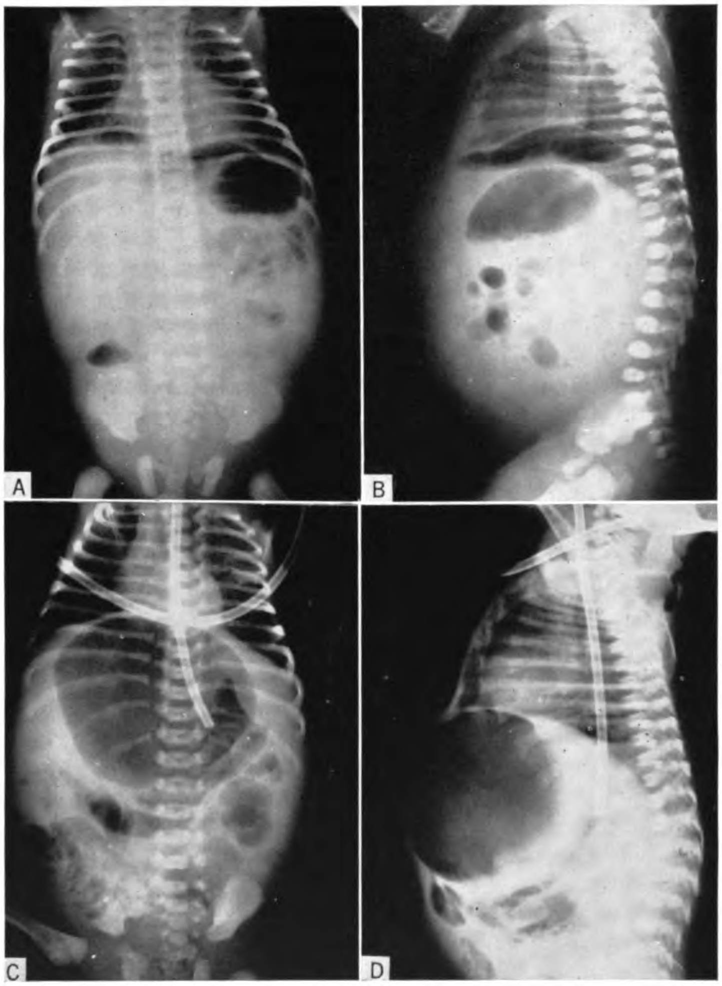

FIG. 27.—*A*, Pneumoperitoneum in a 6-hour-old infant with a double rotation volvulus of the ileum with perforation and peritonitis. At operation gangrenous ileum was resected.

B, Same patient, erect lateral film showing gas under the diaphragm.

C, After operation there was leakage from the side to side anastomosis, forming a large mid-abdominal air bubble.

D, Same patient, lateral film. At necropsy adhesions formed a large cyst-like space containing bile-stained fluid.

tion of the distal colon may be associated with nonrotation of the entire intestinal tract and a common mesentery. In these instances a high midline position of the cecum is encountered. Kleitsch and Reiser noted that complete nonrotation of the bowel may be associated with torsion, resulting in colonic obstruction, and reported such a case in a 31-year-old man. This patient had had several operations for intestinal obstruction believed due to adhesions from a previous appendectomy. A barium enema examination showed obstruction at the mid-transverse colon. Operation revealed complete nonrotation of the bowel, with the cecum and ascending colon on a long mesentery. These had rotated at the midcolon, producing obstruction. Brown and Ross mentioned sigmoid dolichocolon with obstruction due to volvulus in 4 young adults.

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VOLVULUS

Gastric Volvulus.—This is predicated upon faulty mesenteric attachments with elongated gastrohepatic, gastrocolic and gastrolienal ligaments, associated with recent weight loss in obese patients, trauma, eventration of the diaphragm, or with esophageal hiatus hernia. While seen mainly in adults, it has also been observed in children. Two principal forms are recognized. In the more frequent organo-axial type the stomach rotates upwards along its long axis in the coronal plane with the cardia as the fixed point. The greater curvature rolls anteriorly over the lesser, inserting itself between the cardia and the duodenum. With mesentero-axial volvulus the stomach rotates from right to left or left to right about the long axis of the gastrohepatic omentum.

Organo-axial volvulus is characterized by a cascade-like configuration of the stomach, the greater curvature superior to the lesser curvature. Sometimes an hour-glass configuration of the pars media with a spiral configuration of the rugal folds is present. The pyloric ring and duodenal bulb are inclined downwards and to the right, increasing the relative width of the duodenal loop and elongating the second and third portions of the duodenum.

In mesentero-axial volvulus the lesser omentum acts as the axis, and the stomach turns at right angles either to the right or left of the gastrocolic ligament. There appears to be no difference in symptoms in the two varieties of gastric volvulus.

Gastric volvulus often is an intermittent and innocuous abnormality. At times the deformity is very evident, and later vanishes completely. Occasionally a chronic volvulus is associated with minor gastrointestinal complaints suggesting a chronic peptic ulcer. Infrequently pain in the left chest appears, and if the stomach becomes dilated dyspnea and a sense of substernal oppression are noted.

Acute gastric volvulus constitutes a surgical emergency when the stomach becomes incarcerated and strangulated, with consequent necrosis and perforation. There may be a sudden onset of increasing epigastric pain, fullness, nausea and inability to vomit. Scout abdominal films show the stomach twisted upon itself with one and sometimes two fluid levels in the upper abdomen. Since gas cannot pass from the stomach into the small intestine, no distension is present unless ileus develops later. Severe gastric dilatation can cause sudden death because of its compressive effect on the heart.

Strangulation of the stomach in a hiatal hernia can simulate gastric volvulus. The identification of two fluid levels in the retrocardiac and paracardiac areas indicates gas and fluid within a stomach which has rotated itself so that two non-communicating pouches are formed.

In some cases of gastric volvulus the posi-

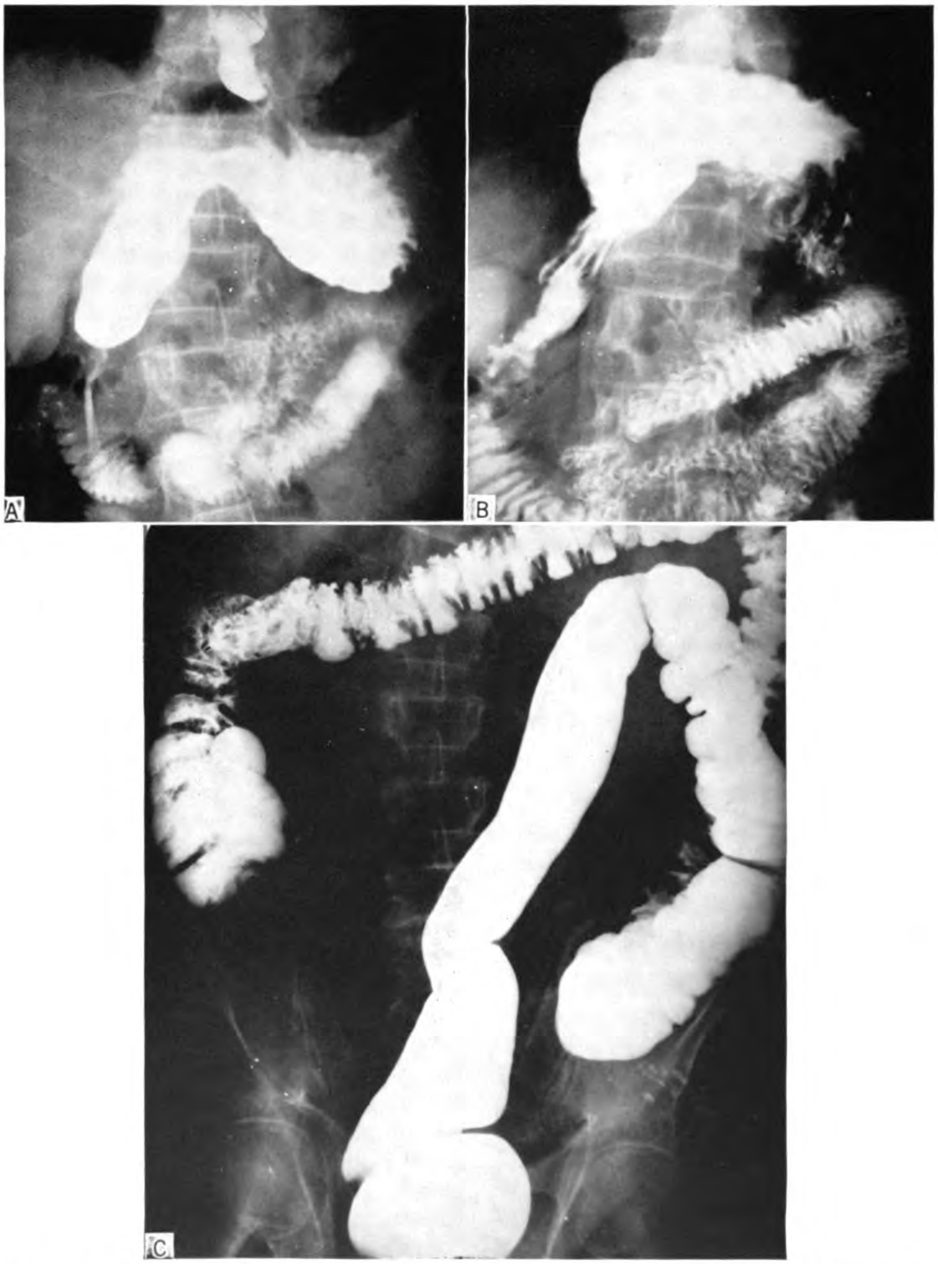

FIG. 28.—*A*, Erect stomach roentgenogram showing organo-axial volvulus in a 58-year-old man with long-standing abdominal distress.

B, Same patient, prone stomach roentgenogram showing persistence of the gastric volvulus.

C, Barium enema examination of same patient revealing dolichocolon. At operation the volvulus of the stomach was no longer present. Partial sigmoidectomy was performed and the patient was relieved of his complaints.

tion and patency of the cardio-esophageal junction is altered. This is best checked by means of a barium bolus. Difficulty in distinguishing between a cascade stomach and gastric volvulus sometimes is resolved by erect frontal and lateral films which help differentiate a normally situated pars media and antrum from one rotated into an abnormal situation.

One of our cases, a 57-year-old man, had longstanding abdominal distress attributed to organo-axial volvulus (Fig. 28). Because of the constant discomfort, surgery was advised. A barium enema done before operation disclosed a dolichocolon, with the sigmoid high in the left upper abdomen. At operation the gastric volvulus was no longer present, and no gastric deformity was observed. The sigmoid was so redundant that it was decided to shorten it by partial sigmoidectomy. The patient's complaints were greatly relieved.

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Volvulus of the Large Intestine.—The most common sites of volvulus of the large bowel are the sigmoid and the cecum. This disorder is predicated on the presence of an unusually long mesentery, the narrow base of its attachment acting as a fixed point about which the bowel rotates. Usually a rotation of 360 degrees is required to produce obstructive symptoms. However, a rotation of 180 degrees also may be significant if the blood supply to the involved loops is impaired.

Sigmoid volvulus is the more frequent form, and often is associated with colonic obstruction of the closed loop variety. The cause for the twisting of the redundant bowel is believed to be changes in the quantity of bowel content together with an abnormal point of fixation. The rectal limb of the loop distends first, and as it dilates it passes upwards and crosses anterior to the proximal or colonic limb. Outpouring of gas and fluid into this loop gradually, or sometimes quite rapidly, produces marked distension, cramps nausea, vomiting and obstipation. As the obstruction persists, vascular impairment and strangulation occur with ultimate perforation and peritonitis. The distention of the bowel involved often reaches enormous proportions, with relatively little dilatation of the remainder of the colon or of the small intestine. However, when strangulation occurs and intraperitoneal exudation of blood and fluid takes place reflex ileus appears. Sigmoid volvulus can resolve spontaneously, and become recurrent as occurred in 2 of our patients who had authenticated episodes 10 and 14 years apart respectively.

The radiologic diagnosis of sigmoid volvulus often is possible from scout roentgenograms which disclose a markedly dilated loop of large bowel springing from the pelvis high into the abdomen. At times the dividing line between the two limbs of distended bowel is sharply seen. With the patient supine, no definite fluid is apparent, but erect studies frequently disclose fluid levels. Sometimes a confirmatory examination with barium enema is necessary. A block is discerned at the point where the rotation of the bowel has taken place, the configuration at this obstructive site assuming a beak-like appearance. The mucosa leading to this

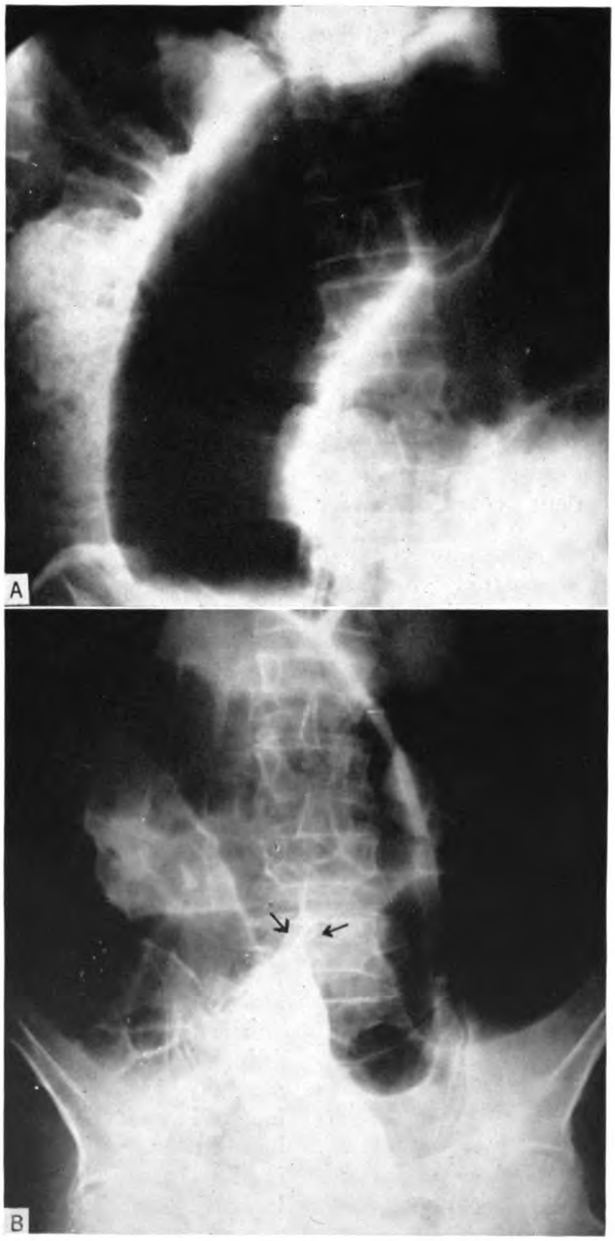

FIG. 29.—*A*, Erect abdominal film of a 75-year-old man with volvulus of the sigmoid. The enormously dilated sigmoid practically fills the abdomen.

B, Same patient. Barium enema reveals a tapered obstruction at the point of rotation of the volvulus (arrows).

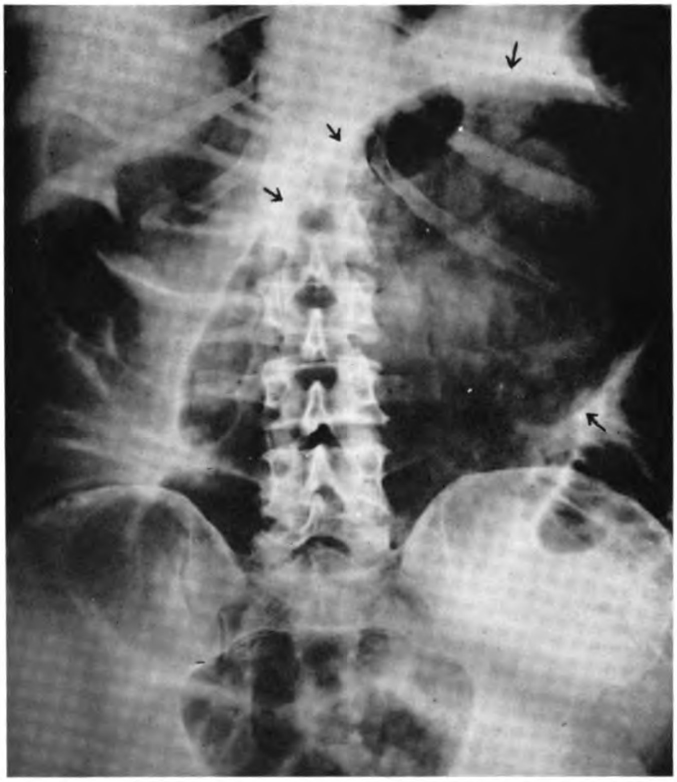

FIG 30.—Cecal volvulus in a 62-year-old man with the enormously dilated cecum reaching high into the left upper abdomen (arrows).

point becomes spiral. A small amount of barium may enter the distended loop, but usually it is better not to force barium further than the beak-like obstruction. If there is a possibility that the bowel wall is impaired, water soluble contrast material is a good substitute for barium.

Volvulus of the cecum and ascending colon is next in frequency. While more frequent in younger people, it occurs in all ages. The mechanism is much the same as sigmoid volvulus, namely a long band-like mesentery with pedunculated fixation of the cecum to the posterior abdominal wall. There is a tendency for abnormal location of the cecum, which often is found in the left upper quadrant. When only partial torsion of the cecum exists, incomplete obstruction permits passage of gas into the distal colon. A partial rotation of the right colon with incomplete obstruction may be suspected when roentgenograms reveal progressive cecal dilatation with a fluid level and apparent discontinuity of the gas pattern in the proximal colon near the hepatic flexure.

The causes of sigmoid and cecal volvulus are much the same. Overeating, overexertion, constipation and heavy purgation are known to precipitate this condition. It is also observed in postoperative patients who become distended, and has been reported in association with abdominal tumors, mesenteric cysts, foreign bodies, chronic mesenteric adenitis and pregnancy. In a report of 8 cases of cecal volvulus, Wilson and his colleagues commented on the importance of lesions of the left colon predisposing to this condition. Four of the 8 cases coexisting disease on the left side, 3 neoplastic and 1 a benign stricture. Gerwig noted that the symptoms of sigmoid volvulus simulated large bowel obstruction, while cecal volvulus tended to point towards lesser bowel obstruction.

The characteristic radiographic picture is one with the markedly distended cecum situated in the left upper abdomen as a single, large fluid-air bubble. Spiral distortion of the mucosal folds at the site of twisting is occasionally observed, and the obstructive point can be demonstrated by contrast enema investigation. Variable dilatation of the small intestine with fluid levels suggesting intestinal obstruction are present.

The identification of cecal volvulus is an indication for surgery. As with sigmoid volvulus, the great danger is impairment of the vascular supply of the bowel which can progress to hemorrhage, infarction, gangrene, perforation and peritonitis. Actually the number of turns made by the rotating bowel loop is less important than the degree to which the vascular supply is impaired.

In one of our recent cases the radiologic picture was classic of cecal volvulus, with a large fluid level in the upper abdomen. On barium enema examination an abrupt block was encountered at the splenic flexure, and it was assumed that the cecal volvulus was dependent on a colonic obstruction due to neoplasm. At operation it was found that the splenic flexure had herniated through a rent in the mesentery, and had rotated upon itself producing an obstruction. Secondary thereto the cecum had become markedly distended, and being on a long mesentery, had rotated upon itself and come to lie in the left upper quadrant.

Volvulus of the transverse colon is uncommon. Cases have been recorded of patients with extremely redundant transverse colons whose flexures were in unusually close proximity. The torsion mechanism in these is similar to that of sigmoid volvulus. The presence of an obstruction in the descending and sigmoid colon may induce rotation of the transverse colon, particularly if adhesions fix a point of the colon to the abdominal wall to provide a pivotal point.

Volvulus of the splenic flexure is even less common. Buenger reported 2 cases he believed to be the first on record. The clinical course was similar to volvulus elsewhere, with chronic intermittent complaints of moderate severity. He believed that partial volvulus of the splenic flexure might be an obscure cause of chronic bloating, constipation and gurgling. From his second case it might be assumed that the condition can be reduced during barium enema examination and thus go unrecognized. Otherwise, the radiologic findings are similar to sigmoid volvulus.

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HEPATODIAPHRAGMATIC INTERPOSITION

Because the anterosuperior surface of the liver has no ligamentous attachment, it is possible for a loop of intestine to enter the space between the liver and the diaphragm. In our experience this is a relatively asymptomatic condition seen most often in adults and only occasionally in children. Torgerson observed that hepatodiaphragmatic interposition was relatively frequent in psychiatric hospitals, and was associated to some degree with gastroduodenal complaints and cecal volvulus.

Radiologically hepatodiaphragmatic interposition is usualy identified on routine chest roentgenograms by the presence of an air containing loop of bowel below the right diaphragm. Usually this is colon, but occasionally small intestine enters the area. The nature of the loop is best identified on gastrointestinal examination. Infrequently some difficulty in distinguishing between hepatodiaphragmatic interposition, pneumoperitoneum or subphrenic abscess may arise, but this can often be resolved on clinical evidence. Nevertheless, the possibility of the coexistence of two disorders must not be overlooked.

Rarely hepatodiaphragmatic interpositions become fixed because of inflammatory adhesions. If symptoms so indicate, operative reduction of this malposition is desirable.

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INTRA-ABDOMINAL HERNIAS

An important consequence of intestinal malrotation is disarrangement of the mesenteric attachments predisposing to internal hernias. Among these are hernias into the lesser omental sac through the foramen of Winslow, paraduodenal hernias, pericecal hernias, hernias into the intrasigmoid area and herniations through defects in the mesentery, the omentum and the broad ligament. Except for the latter, all are retroperitoneal hernias. These may occur in any age group from infancy to old age.

Intra-abdominal hernias causing intestinal obstruction manifest themselves by recurrent abdominal distress and pain, nausea and distension. During severe attacks the nausea and abdominal discomfort often are intense. Vomiting, however, is relatively infrequent. The clinical indications of intra-abdominal inflammations are absent. Symptoms are intermittent, and their durations varies from a few days to weeks or even years. With vascular impairment symptoms become more intense, and hemorrhage takes place if the involved bowel loop is infarcted.

The radiologic pattern of the various internal hernias are relatively similar. Often one can only suggest this possibility, without being specific as to its nature. The radiologic indications are dependent on the displacement or disturbance of arrangement of the small intestine, characterized by clumping of the coils and stasis proximal to or at the hernial site. The extent of a hernia can vary from involvement of only a small segment to incarceration of most of the lesser bowel. Instances have been reported in which the gallbladder, the omentum, the stomach and the large intestine also have been involved.

Paraduodenal Hernias.—These are relatively infrequent, and occur more often on the left than on the right side. A loop of small bowel may enter any one of nine separate orifices. The most frequent sites are in the upper and lower portions of the fossæ formed by the ligament of Treitz. With right paraduodenal hernias the invaginated small bowel is caught in a peritoneal sac behind the ascending and transverse mesocolon. The opening of this sac is near the duodenojejunal junction at the ligament of Treitz,

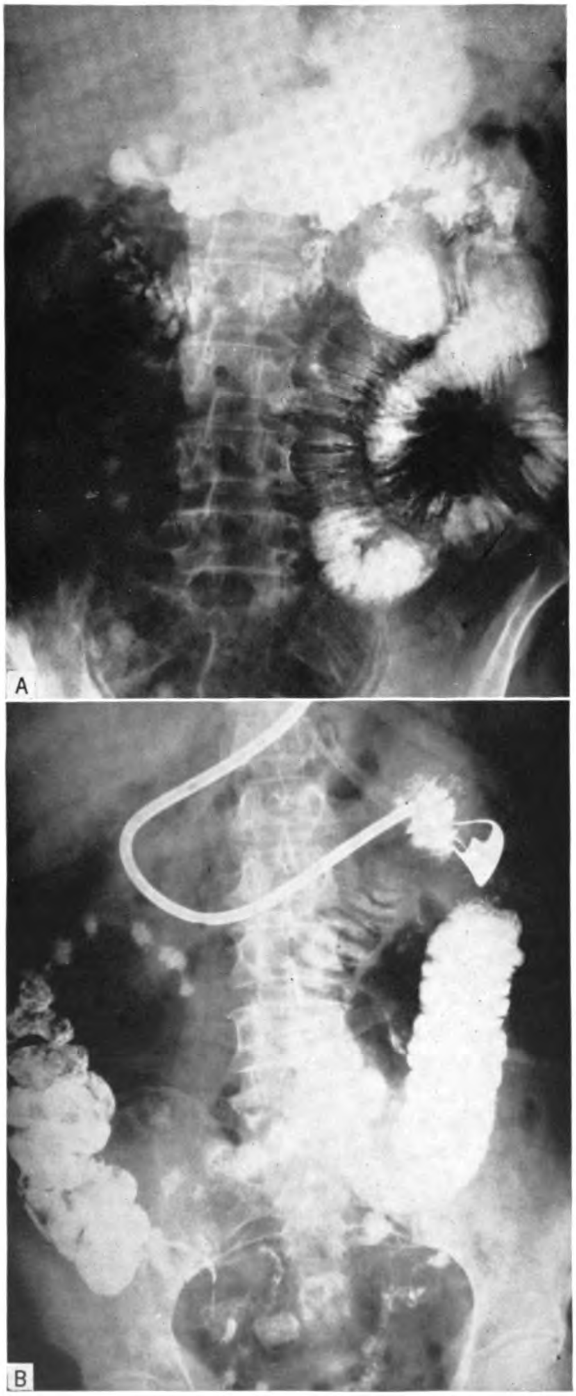

FIG. 31.—*A*, Left paraduodenal hernia in a 65-year-old man with high intestinal obstruction. **Note the** distended jejunal loops in the left midabdomen.

B, Same patient 24 hours later. Barium is still present in the dilated jejunum. At operation a **jejunal loop 5 feet from** the ligament of Treitz had herniated through the paraduodenal fossa.

and the superior mesenteric artery lies in the anterior portion of the sac. The orifice faces to the left side, while the blind extremity is directed to the right and downwards. The appearance of the small intestine in paraduodenal hernias is such that the affected bowel appears confined in a spherical transparent bag which restricts position and movement by manipulation or changes in posture.

In most reported cases the diagnosis was unsuspected before operation. Reeves, Moran and Jones reported that only 2 out of 51 cases in the literature were correctly diagnosed. They reported a third in which a correct diagnosis was established because of the spherical appearance of the small bowel loops suggesting a sac-like structure around which the colon was displaced. Some constriction of the duodenum at the neck of the sac was shown in their illustrations. The fact that correct diagnosis may be made more often was suggested by Parsons, who gathered 12 cases from his own experience in which paraduodenal hernias were diagnosed radiologically. In 8 cases the small intestine was found incarcerated at operation, and in the remaining 4 no entrapment was found. Parsons considered this due to spontaneous reduction of the lesions.

With left paraduodenal hernias the stomach is superior to the sac, and a clear space exists between it and the stomach. The ascending colon remains to the right, whereas with right paraduodenal hernias the ascending colon is posterior. Hernias adjacent to the ligament of Treitz displace the proximal jejunum upwards or downwards and medially. The displaced intestinal loops are represented by a clump of gas-filled relatively fixed shadows. Fluoroscopic inspection is important in identifying these atypically situated intestinal loops. Whenever paraduodenal hernia is suspected, the patient should be examined at frequent intervals immediately after the barium meal. When no obstruction exists the diagnosis may be overlooked if particular attention is not given to the fluoroscopic as well as the roentgenographic observations. The differential diagnosis between right and left paraduodenal hernia is aided by the fact that the herniated loops are situated either to the right or left of the midline respectively.

Herniations through the *foramen of Winslow* are uncommon, and are associated with mesenteric abnormalities such as poor fixation of the right colon or an abnormally long mesentery of the small intestine. Other factors include an abnormally large foramen of Winslow, increased intra-abdominal tension, a low projecting right lobe of the liver with a concave inner aspect resulting in a tendency for the gut to be funneled cephalad. Sometimes no predisposing cause is found. Either the small or the large intestine enters the lesser omental sac, and rarely the gallbladder finds its way into this space. Symptoms usually are chronic, with vague gastrointestinal disturbances for years. Instances of acute obstruction of incarcerated bowel have been reported.

The correct diagnosis may be made radiologically by identifying a radiolucent fluid-containing collection medial to the stomach, displacing it anteriorly. Hernias into the lesser omental sac also may occur between the stomach and the spleen through the gastrolienal ligament, displacing the stomach anteriorly and the spleen posteriorly. The left lienorenal ligament also may permit herniation, displacing the spleen anteriorly and laterally. The presence of gas and fluid containing intestinal loops either posterior to the stomach or displacing the spleen suggests the presence of a hernia into the lesser omental sac.

It is possible for a large amount of bowel to enter the hernial sac. If colon enters, a barium enema may disclose a tapering block at the hernial site pointing towards the foramen of Winslow. A case in which the cecum, ascending colon and hepatic flexure had herniated into the lesser sac was reported by Dorian and Stein. The correct diagnosis was suggested by an unusual radiolucency with a fluid-air level immediately medial to the stomach. While an air-fluid level in the lesser sac also may be caused by a perforated ulcer, small multiple levels and small bowel markings constitute an almost specified sign of herniation into the lesser omental sac.

Small intestinal incarcerations are occasionally found in the *pericecal areas*. Radiologically these hernias simulate obstructive lesions, a specific diagnosis rarely being made. Recently we saw a 5-year-old boy with intermittent, severe cramps unrelieved by medication for about a month. Radiologic examination showed clumped coils of small intestine in the right lower quadrant. At 6 hours a considerable amount of barium remained proximal to these clumped loops, and at 24 hours the lesser bowel was empty. This was reported as a partially obstructive extrinsic lesion, possibly an internal hernia. At operation the midileum was found to be pulled into a pocket in the right lower quadrant made up by Jackson's membrane.

Herniations also may occur occasionally in the region of the sigmoid in the fossa between the mesosigmoid and the parietal peritoneum, the fossa of Waldeyer. Entrance of small intestine into this fossa produces partial intestinal obstruction.

Transmesenteric hernias constitute about 11 per cent of all intra-abdominal hernias, and occur in any age group. These consist of protrusion of a knuckle or loop of bowel through a hole in the mesentery, and lead to intestinal obstruction and strangulation. In the 50 cases described by Cutler and Scott, the defect was at the ileocecal junction in 35, in the midileum in 6 and in the jejunal mesentery in 2. One occurred in the mesosigmoid, and another had multiple fenestrations. Usually the holes are rather small, averaging 2 to 3 cm. in diameter. These are circular or oval in shape with smooth peritonealized edges. Much larger openings may be present.

The clinical picture of *transmesenteric herniation* is one of intermittent cramps, vomiting, distension, pain and malaise. With strangulation acute symptoms may appear. Radiologically the changes are those of intestinal obstruction. Rarely an incarcerated loop of bowel is identified, but usually the diagnosis is made at operation. Of our 2 verified cases, 1 was a child 8 weeks old with a clinical picture of volvulus and radiographic findings of intestinal obstruction. The second was a 65-year-old man with large bowel obstruction and cecal volvulus. The bowel obstruction was due to herniation of the splenic flexure through a rent in the mesentery.

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CONGENITAL BANDS

Errors of development of the mesentery also result in thickened folds or web-like strands variously described as bands, ligaments, membranes, or congenital adhesions. In the upper abdomen these often extend from the duodenum to the liver or colon, and either cross or are attached to the first and second portions of the duodenum. The duodenal bulb is sometimes deformed, and the second portion may be pulled outwards and to the right at varying angles. A high fixation or deformity of the bulb often is more apparent on erect roentgenograms.

Although congenital webs or bands often

produce no symptoms, they may cause intestinal obstruction. In infants obstruction of the duodenum is seen together with volvulus of the small intestine and malposition of the cecum. In 2 cases reported by Dolin, Mathews and Russo concomitant volvulus and pressure by an abnormally situated and distended cecum contributed to the obstruction. In older patients the clinical picture simulates gallbladder disease or duodenal ulcer. The diagnostic difficulties are increased when such bands produce irregularities of the duodenal bulb with stasis and pylorospasm with gastric retention. Infrequently, congenital as well as inflammatory bands deform the pylorus so that it is not possible to differentiate this condition from neoplastic infiltration. Neither can congenital bands be differentiated radiologically from inflammatory adhesions. Jenkinson, Pfisterer, Norman and Latteier reported 7 patients, all over 50 years old, with symptoms of gastrointestinal bleeding and rigidity and narrowing of the antrum with such changes.

Obstruction of the large bowel by congenital bands is seen occasionally. Safran and Greenberg described a 12-year-old girl with abdominal pain, nausea and vomiting with an atypical defect in the transverse colon. At operation a congenital fold of peritoneum extending from the aorta to the colon was found. Overton, Bolton and Usher also reported such deformities mimicking carcinoma. We observed a 5½-year-old girl with intermittent abdominal cramps and occasional vomiting for 2 years. She had been operated upon a year before for a malrotated cecum and ascending colon, with no relief after reposition of the cecum. Gastrointestinal examination showed no pathologic change, but on barium enema the sigmoid was seen fixed in position and the splenic flexure was depressed. A congenital band fixing the descending and sigmoid colon causing partial obstruction was diagnosed. At operation the lowermost part of the descending colon and adjacent sigmoid were encircled by a thick band which extended from the root of the mesentery across the intrapelvic descending colon, and inserted near the left fallopian tube. Relief followed resection of this band.

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CONGENITAL GASTROINTESTINAL DIVERTICULA

True congenital gastric diverticula are outpouchings of all the layers of the gastric wall. False diverticula follow inflammatory lesions, perigastric adhesions, foreign bodies, gastric ulcers with perforation, or ulcerating tumors. Extrinsic factors such as inflammations of the gallbladder, pancreas, spleen or regional lymph nodes also can produce false diverticula.

Gastric diverticula are quite frequent, occurring in about 2 per cent of cases. They usually appear on the posterior aspect of the lesser curvature just below the cardioesophageal junction. Among predisposing factors are included congenital weakness of the gastric musculature permitting protrusion of mucosa because of increased intragastric pressure and strain, weakness at the site of passage of large blood vessels through

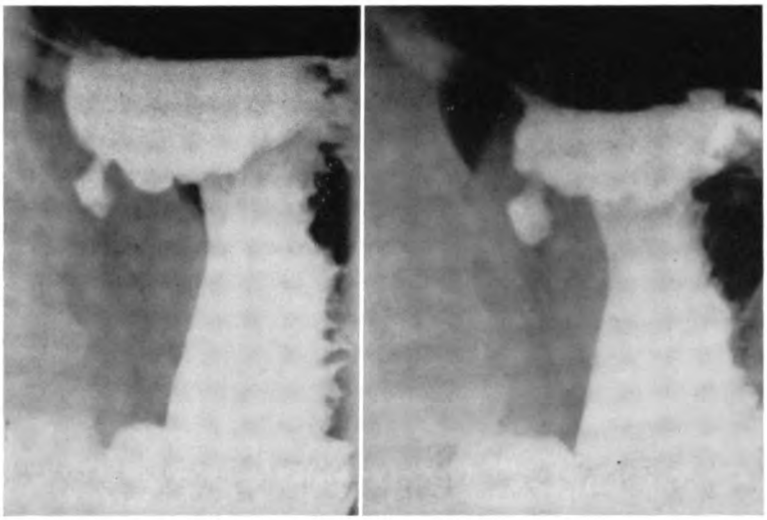

FIG. 32.—Gastric diverticulum high on lesser curvature of stomach. This could not be found by the surgeon at operation, and was again demonstrated on a repeat examination.

the gastric wall, and weakness in the peritoneum as it is reflected in the formation of the gastrosplenic and lienorenal ligaments.

Gastric diverticula vary in size from about 1 cm. to more than 9 cm. in diameter, with considerable variation in the size of their openings into the stomach. Occasionally aberrant pancreatic tissue is present. As a rule, no symptoms are produced, but there are occasionally acute disturbances. Moses reported a case with rupture of a gastric diverticulum and intraperitoneal hemorrhage. Ogur and Kolarsick reported 2 cases of gastric diverticula in infants, one with intussusception of a true diverticulum into the stomach causing gangrene, perforation and peritonitis, and the other with a false diverticulum believed to be secondary to pyloric stenosis. Hemorrhage occurred in 2 out of 30 cases reported by Brown, Bissonnette and Albee.

The radiologic diagnosis is made often by closely observing the passage of barium into the stomach after the patient has swallowed the first mouthfull. The entrance of the contrast material into the pouch is best observed in the oblique position. Occasionally a diverticulum missed on the original examination can be detected by persistence of barium within the pouch on later films. The deceptiveness of this lesion is brought out by the fact that on two occasions when operation was deemed necessary the surgeon was unable to find the diverticulum, only to have it clearly demonstrated on radiologic examination soon after operation.

Duodenal diverticula are commonly located on the concave side of the duodenum near the ampulla of Vater, but may arise from other positions in the duodenal loop. Most often they are solitary, but bilobed or trilobed pouches are not uncommon. True duodenal diverticula contain all the duodenal layers, while false ones, as elsewhere, represent herniations of mucosa through defects in the muscularis. The clinical incidence has been reported as about 2 per cent, but at necropsy about a 10 to 15 per cent rate of occurrence is present.

Usually duodenal diverticula are asymptomatic, but bizarre incidents have been reported. Blegen, Swanberg and Cox reported a verified case, apparently the seventh, in which the common bile duct entered a duodenal diverticulum. Some duodenal diverticula contain aberrant gastric or pancreatic tissue, predisposing to ulceration with pain, local tenderness and hemorrhage. Rarely extrinsic pressure against the common bile duct or duodenum may cause jaundice, in-

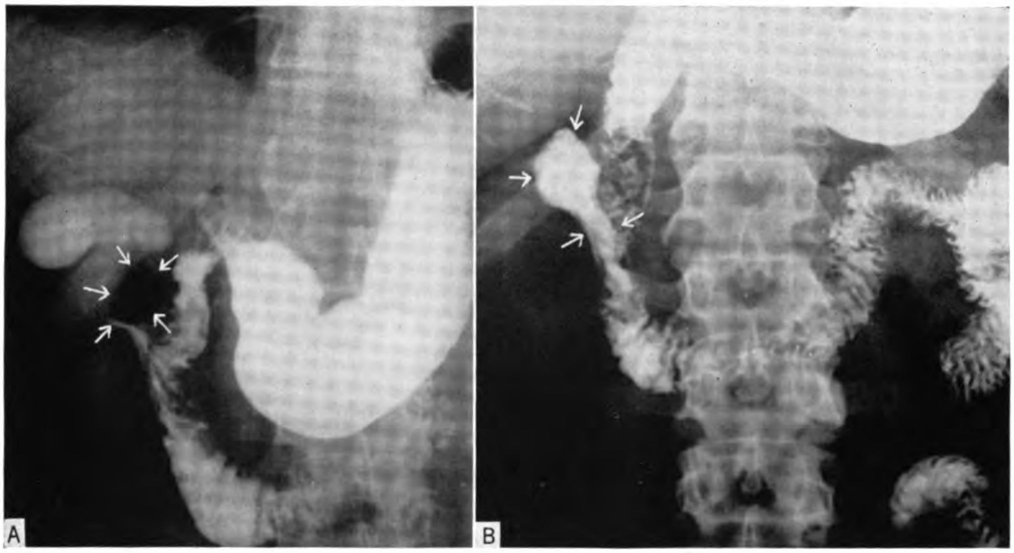

FIG. 33*A*.—Atypical duodenal diverticulum, asymptomatic. The pouch arises from the inner aspect of the midportion of the descending duodenum and passes laterally and upwards. In the erect position the pouch is filled with gas (arrows).

B, Same patient, supine film showing elongated diverticulum filled with barium (arrows). Note the change in the position of the diverticulum in relation to the gallbladder.

termittent duodenal stasis or obstruction. Another rare occurrence is tumor formation within the pouch, as reported by Dunstan, Lowance and Jones.

The radiologic diagnosis of duodenal diverticula is relatively easy. These structures vary in size from 1 cm. up to 5 or 6 cm. in diameter. Most are relatively fixed in position. Some have long necks, and come to rest behind the pylorus. Frequently horizontal streakings of mucous membrane leading into the pocket can be identified. Fluid levels appear in some diverticula with the patient erect, and retention of barium for several hours is not uncommon.

Diverticula occur fairly often in the *small intestine*, particularly the jejunum, and as a rule are asymptomatic. Congenital diverticula tend to form on the antimesenteric wall, while acquired diverticula are close to the mesenteric border. There appears to be a tendency for multiplicity in jejunal diverticula. In our own files are 3 cases with multiple jejunal diverticula, none of which were regarded as clinically significant. Nevertheless, when inflamed, small intestinal diverticula may simulate acute appendicitis, and complications such as intestinal obstruction and rupture have been reported. The presence of aberrant pancreatic or gastric tissue also predisposes to ulceration and hemorrhage.

Gross, Neuhauser and Longino reported cases in which long thin diverticula arose from the duodenum or jejunum, penetrated the right diaphragm and entered the thorax. These diverticula extended upwards in an extrapleural position, usually in the posterior chest, and assumed a variety of configurations. Symptoms were produced by accumulations of gas and fluid in the thoracic portions of the diverticula and from ulcerations and hemorrhage. The walls of these sacs contained smooth muscle as well as mucosa resembling stomach in 2 cases and jejunum in 1 case. Radiographic examination disclosed air filled loops due to coiled diverticula, the mass extending out from the mediastinum towards the right.

Congenital diverticula of the *colon* are more frequent in the cecum. These occur more often in adults than in children, and

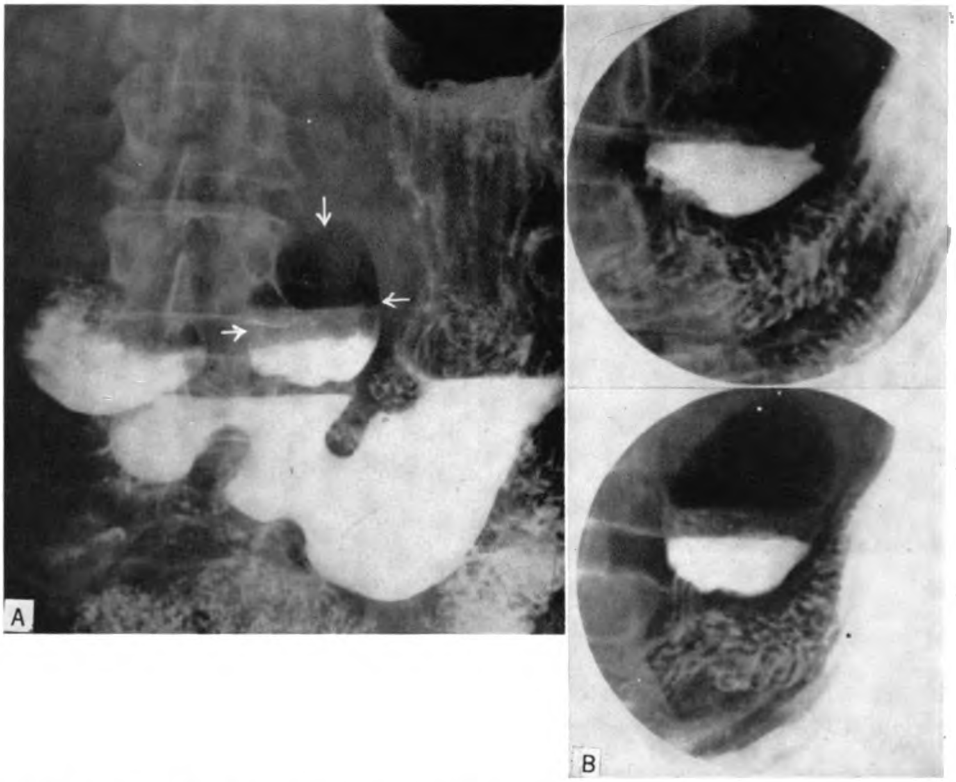

FIG. 34.—*A*, Large fixed jejunal diverticulum above the lesser curvature of the stomach seen on erect film *B*, Same patient, spot compression studies reveal the mucosal folds leading into the diverticulum.

sometimes are accompanied by Meckel's diverticula. When uncomplicated, these sacs are readily recognized radiographically. With supervening inflammation, cecal deformities indistinguishable from carcinoma appear. Cecal diverticulitis with peritonitis and abscess formation simulates acute appendicitis, and usually is discovered at operation.

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DUPLICATIONS OF THE GASTROINTESTINAL TRACT

This term is used by Gross, Holcomb and Farber to include malformations of spherical or tubular structures, with well-developed smooth muscle layers lined with mucous membrane. Duplications of the digestive tract occur at any level from the tongue to the anus. The embryologic basis rests on the fact that in early fetal life the intestinal lumen is completely occluded by proliferations of epithelial cells. With growth, vacuoles appear in this cell mass, which eventually unite to form the normal passage. Some vacuoles fail to communicate, and form their own mucosal and muscular coats. These may have a common wall with the normal bowel, or may be suspended from their own mesenteries. The resultant anomalies are either tubular structures branching from the intestine and extending between the mesenteric leaves, or they may reach the thoracic cavity. When developed as double-barreled structures, communication with the lumen of the parent structure occurs at one or at both ends. Cyst-like duplications often lay intraperitoneally, contiguous with the bowel and attached by a thin mesentery. The mucosal linings are not necessarily similar to that of the adjacent alimentary tube, although the muscular layers may be fused. Ectopic gastric and pancreatic tissue is frequent.

Five major groups of duplications are identified. These include intramural cysts, spherical duplications, tubular duplications, abdominal thoracic duplications and mesenteric cysts with walls which histologically represent intestine. Duplications infrequently affect the gallbladder, producing either completely separate sacs each with a common duct, or with the common ducts joining into a Y-shaped structure with a single opening. With incomplete duplication the gallbladder may be bilobed or septate.

Gastric duplications are uncommon, and

rarely are diagnosed preoperatively. Nolan and Lee reported 2 cases, one associated with hemorrhage. These had duplications elsewhere in the intestinal tract as well. An interesting example of gastric duplication was reported by Barbosa, Decastro and Caminha in a 33-year-old woman with severe epigastric pain, vomiting and a large tender mass in the left upper quadrant dislocating the stomach anteriorly. At operation a large duplication of the stomach containing more than 2 liters of fluid was found.

Enterogenous cysts in the wall of the stomach are also found occasionally. Dewing, Roessel and Olmstead report that they consider the seventeenth such case in which the lesion about 3 cm. in diameter was found incidentally in the fundus of the stomach.

Duplicative cysts of the duodenum occur in all age groups, and become symptomatic when they impinge against adjacent structures causing partial or complete obstruction. Occult or profuse bleeding also has been noted. These cysts originate as diverticular pockets within the wall of the primordial duodenum, and later become an integral part, separated from the main channel by a mucosal-submucosal layer. Since the cyst lining is an isolated secreting mucosal surface, gradual expansion takes place as fluid accumulates. Radiologically, these cause obstructive changes and displacement of the stomach and duodenum. Symptoms often appear in the first year of life, with partial or complete obstruction and hemorrhage.

Duplications of the small intestine are quite frequent in the distal ileum, occurring most often in infants and children. Associated with these may be malposition of the lesser bowel with volvulus, obstruction and hemorrhage. The clinical picture is in relation to the size and location of these lesions, which frequently produce obstruction. Impairment of blood supply is followed by infection, rupture and peritonitis. Communication of small intestinal duplications with intrathoracic extensions has been mentioned. Additional cases of this variety were reported by Caruolo, Ellis and Harris and by Snodgrass. The presence of ectopic gastric and pancreatic tissue again predisposes to ulceration and hemorrhage.

Duplications of the colon occur either as small parallel structures, or are large enough to parallel the entire length of the large bowel resulting in a double colon. They also may simulate large cysts more or less alongside the bowel within the mesentery. There also may be 2 terminal ileums and 2 appendices. A case of duplication of the entire colon with no communication either with the bowel or the outside was reported by Weber and Dixon. Partial duplications occur at any point along the wall of the bowel. These may end blindly, resulting in cyst-like structures. Such a cyst with its own mesocolon was reported in a 10-year-old girl by Gondos.

Twinning of the hindgut anlage results in duplications of the bladder and urethra. This smaller group is usually asymptomatic except when errors such as imperforate anus, double rectum or imperfect formation of the septum between the rectum and the urogenital sinus occurs. An interesting case of duplication of the rectum was reported by Cogswell and Thompson in a 7-week-old female infant. This presented as a large air-containing sac which displaced the abdominal contents to the right. No communication could be demonstrated between the colon and this cyst. The presence of air was postulated on the presence of a ball-valve mechanism.

Intrathoracic cysts sometimes are associated with congenital defects in the cervical and thoracic vertebræ. The embryologic basis for this neurenteric communication is embryonal ecto-entodermal adhesion of the alimentary canal from esophagus to ileum through a cleft notochord to the neural tube during the presomite stage. As development proceeds, this ecto-entodermal connection may form traction diverticula, cysts or neurenteric strands along the line of attachment. Diverticula may occur at either or both ends of this attachment, forming intra-abdominal or intrathoracic cysts distant to, but connected by a strand, to the gut.

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INTRA-ABDOMINAL CYSTS

Intra-abdominal cysts formed by persistence of the middle of the *omphalomesenteric* duct have been discussed before. Primary abdominal cysts, which are relatively uncommon, occur in the omentum, the mesentery or in the retroperitoneal area. These are attributed to obstruction of existing lymphatic channels or by growth of congenitally misplaced lymphatic tissue which fails to communicate with the vascular system. Hinshaw reported that but 174 such cysts had been found in more than 1 million patients at the Mayo Clinic. Mesenteric cysts tend to occur more often in children up to 10 years of age, but are found in all age groups. The mesentery of the small intestine contains about half the reported cases, the remainder being found in the omentum, the mesocolon, the retroperitoneal area or free in the abdominal cavity in rare instances. Multiple cysts are relatively uncommon. Intra-abdominal cysts may vary from only a few centimeters in diameter to a capacity of several liters. They are thin walled, with occasional calcific plaques and are dumbbell-shaped, oval, unilocular or multilocular. While smooth muscle bundles are present in the cyst walls, no true muscular coat is present. This is in contradistinction to congenital duplications of the bowel. The encysted fluid is either serous or chylous, and in some a pasty, putty-like material has been found.

These lesions are identified radiologically by a relatively opaque, sharply-circumscribed intra-abdominal mass which may displace the adjacent viscera. They can be differentiated from dermoid cysts, which usually contain radiolucent fatty material and often bone or teeth. Pancreatic cysts may be identified by their position in relation to the stomach and duodenum.

Occasionally an infant is seen with a lower abdominal mass in whom urography discloses a nonfunctioning kidney on that side. This may prove to be a unilateral multicystic kidney. These cysts are attributed to failure of union between the secretory and collecting systems. As they enlarge, the increased pressure destroys renal tissue, which sometimes goes on to renal failure.

Another infrequent pelvic mass is *hydrometrocolpos* due to an imperforate hymen or atresia of the lower vagina. The marked dilatation of the vagina may extend up to involve the uterine cavity. If sufficiently large, these collections produce intestinal obstruction by extrinsic pressure, as well as symptoms of urinary retention. If symptoms do not appear during infancy, they may be delayed until puberty when amenorrhea becomes evident. Radiologically these appear as homogeneous masses in the lower midabdomen. Occasionally a waist corresponding to the junction of the vagina and uterus is visible.

Simple retroperitoneal cysts, like intraabdominal cysts, are attributed to obstruction of existing lymphatics, or more likely, to congenitally misplaced lymphatic tissue which fails to communicate with the vascular system. The cysts contain clear serous fluid or chylous milky material. A particularly large cyst may be difficult to distinguish from ascites. These cysts appear as gradually painless enlargement of the abdomen, with few symptoms. Occasionally acute pain is produced by torsion of a cyst or by hemorrhage after trauma. Ileus associated with this can mimic intestinal obstruction.

Choledochal cysts form because of congenitally unequal endothelial proliferation at the time of physiologic occlusion of the embryonic common bile duct, resulting in later localized enlargements at the time of recanalization. Congenital weakness of the duct, irregularity of its lumen and obstruction are additional factors. Choledochal cysts occur in any age group, but are more frequent in women under 25 years of age. An enormous variation in size exists, some containing only a few drops while others contain as much as 8 liters of fluid. The cysts usually are unilocular and thin walled, but considerable thickening can be present. Large choledochal cysts appear as upper abdominal masses, with pain and jaundice which may be intermittent if pressure against the common duct is inconstant. Nausea and vomiting often are present. Spontaneous or traumatic perforation has been observed occasionally, with consequent biliary peritonitis.

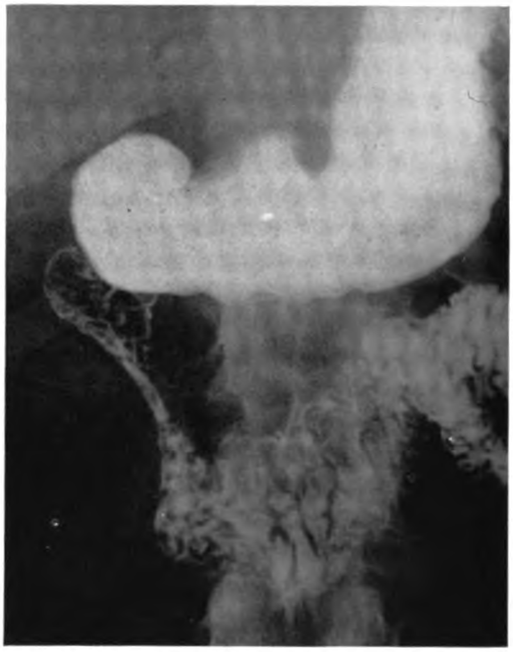

FIG. 35.—Renal cyst impressing lateral aspect of descending duodenum in a 60-year-old man.

Cholecystography usually discloses only non-visualization of the gallbladder, although an occasional case with some opacification is encountered. Intravenous cholangiography may outline the cyst if jaundice is not present. The adjacent viscera often is displaced by large cysts.

Liver cysts occur as mesenchymal hamartomas which appear as symptomless masses in the upper abdomen of children, often before the age of 2 years. These are somewhat more frequent in females. They increase rapidly in size as fluid accumulates. These

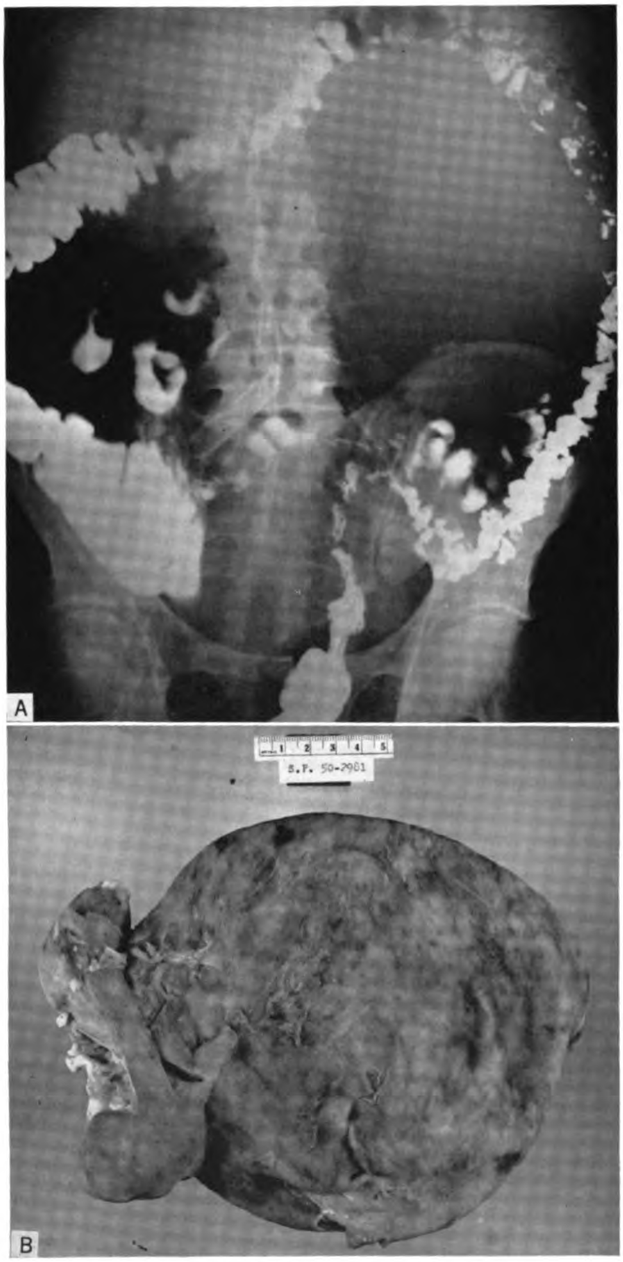

FIG. 36.—*A*, Advanced hydronephrosis of left kidney displacing the splenic flexure in a 78-year-old man. *B*, Specimen, left kidney.

are connective tissue cysts in and around the remnants of portal triads, and contain serous fluid. Coexistent cysts of the pancreas and kidneys may be present. When the cyst wall calcifies, it becomes visible on plain film examination of the abdomen. Otherwise the cysts appear as masses in the right upper abdomen close to the liver. Twisting of the pedicle, hemorrhage or infection within a liver cyst may precipitate acute abdominal symptoms.

Splenic cysts appear radiologically as calcific rings in the left subphrenic area. Lustok and Baum recently reviewed 31 such cases. As a rule no acute symptoms are present. We have seen 3 such cases. One, a 17-year-old girl, had had pain in the left upper abdomen and progressive swelling of this area for about 2 years. A large mass was felt in the left hypochondrium. Diagnostic pneumoperitoneum disclosed a cystic mass with calcium in its periphery. At operation, the spleen was replaced by a tremendous cyst containing brown, semi-opaque fluid. Inasmuch as no history of trauma could be elicited, it was presumed that the cyst was of congenital origin.

Occasionally a hydronephrotic kidney or a renal cyst simulates an intraperitoneal or retroperitoneal cyst. When sufficiently large, either lesion can displace adjacent viscera, and the true nature of the lesion is found at urologic investigation. A cyst originating in the lower pole of the right kidney can impress itself against the duodenum, displacing it anteriorly and medially. Renal cysts not infrequently are readily palpated in the abdomen, particularly if they originate from the antero-inferior aspect of the kidney.

Congenital pancreatic cysts occur anywhere in the pancreas, attain considerable size and displace the stomach and duodenum. The cyst wall may calcify. In one of our cases such calcification simulated an aneurysm of the abdominal aorta.

Adrenal cysts likewise reach a large size, and occasionally are visualized because of calcification of their walls. Their location is identified by their displacement of the kidney. If of sufficient size, these too displace the adjacent bowel. The clinical pattern is dependent upon the hormonal activity of the cyst as well as its size.

Ovarian cysts are frequent in adults, but may also be observed in infants and children. As a rule these present as intrapelvic masses, which may reach advanced size even in younger patients.

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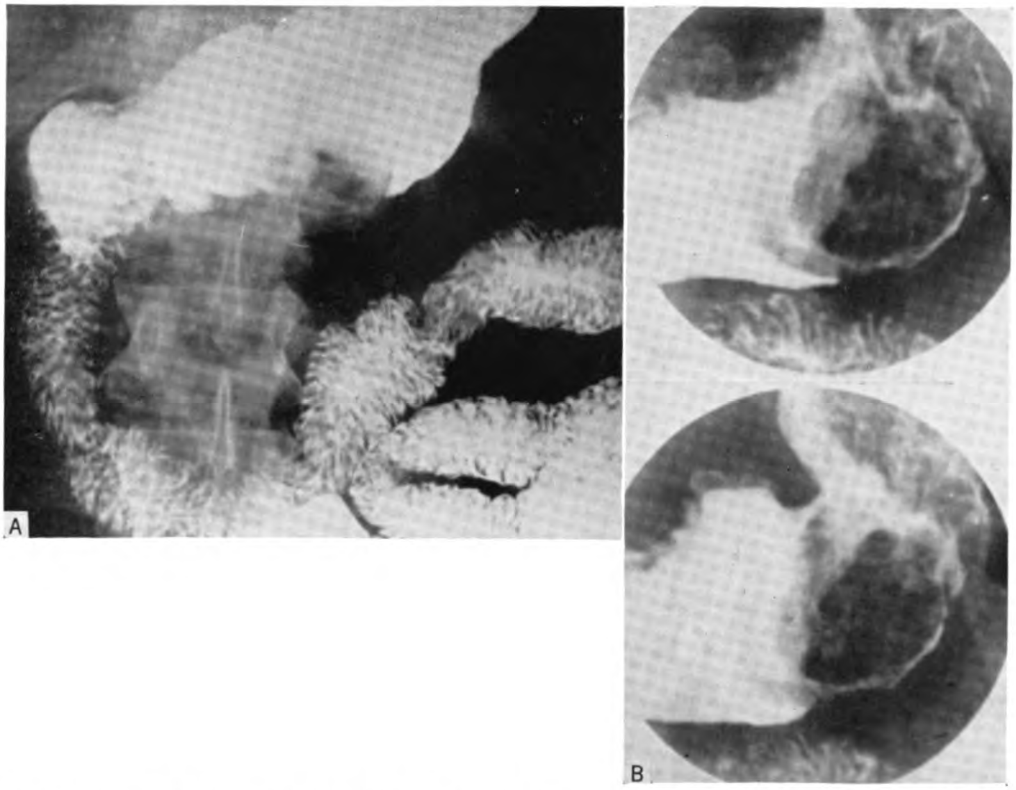

FIG. 37.—*A*, Pancreatic rest along greater curvature of stomach in a 54-year-old male with vague epigastric distress.

B, Same patient, spot compression studies. Microscopic examination of the specimen revealed malignant changes.

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HETEROTOPIC TISSUE IMPLANTS

Heterotopic implants of gastric tissue are frequent in congenital gastrointestinal diverticula and duplications. Ulceration and hemorrhage from these deposits is a prominent cause of complications. Ectopic gastric tissue in the lower esophagus has been associated with obstruction, as described by Thomas, Rappaport and Keogh.

Heterotopic pancreas occurs most often in the stomach, duodenum and jejunum, and also is found in the ileum, the gallbladder, spleen, mesentery, omentum, liver and in congenital diverticula and duplications. This tissue is subject to pancreatitis, malignant degeneration and to islet tissue proliferation with hyperinsulinism. In a review of 410 cases from the literature, Present found 110 with stomach implants, 117 in the duodenum 68 in the jejunum and 21 in the ileum.

In the antrum ectopic pancreatic implants occur as smooth tumor nodules up to about 5 cm. in diameter. As a rule these cannot be distinguished from other mural masses, but occasionally a central umbilication retaining a fleck of barium is a clue to the diagnosis. Duodenal implants occur as nodules which produce irregular elevations as well as circumscribed lesions. Most are less than a centimeter in diameter. Occasionally, heterotopic implants cause intussusception or pyloric obstruction. Mackinnon and Nash reported pyloric obstruction in a 5-year-old child due to heterotopic pancreas. Collett, in a review of 471 cases, found 10 per cent in children, and in 5 the deposit was in the stomach. One had a polyp at the lesser curvature which prolapsed through the duodenum, causing obstruction. Keeley reported the case of a 3½-year-old boy with ileo-ileal and ileo-colic intussusception. The latter was reduced hydrostatically, but the ileo-ileal invagination persisted, and at operation was found to be due to a bit of pancreatic tissue. In a 57-year-old man seen by us, neoplastic changes were found in a submucosal gastric implant of pancreas.

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ANNULAR PANCREAS

Annular pancreas results from failure of the ventral pancreatic anlage to migrate to the right and posterior to the duodenum and to fuse with the dorsal anlage. A tip of the ventral anlage remains anterior, while the duct rotates normally. Thus a band of pancreatic tissue is wrapped around the descending duodenum. The ducts persist in a rudimentary form, and extend from the anterior part of the ring to the left to join the main pancreatic duct. More often the annular pancreas is traversed by a major duct which begins anteriorly, passes to the right and then curves laterally and posteriorly to join the common bile duct or the pancreatic duct. An annular pancreas may vary from a partly encircling non-obstructive lesion to a complete ring with underlying duodenal atresia. In infants it is a cause for high intestinal obstruction. Symptoms also appear later in life, and it is not infrequent for the condition to be asymptomatic and identified only at necropsy. In adults symptoms may simulate peptic ulcer, and such ulcers not infrequently are associated with an annular pancreas. Other congenital malformations such as duodenal bands, partial rotation of the colon and volvulus also occur. Jaundice due to pressure against the bile duct was reported by Anderson and Wapshaw in association with pancreatitis in the annular pancreas.

The radiologic diagnosis of annular pancreas in infancy is suggested by the presence of duodenal obstruction. Marked dilatation of the duodenum above the annular pancreas, together with dilatation and air in the stomach produce a "double-bubble" configuration which is highly suggestive of this entity. In adults the diagnosis is indicated sometimes by a partially indenting ring in the second portion of the duodenum.

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INTRINSIC CONGENITAL GASTROINTESTINAL OBSTRUCTIONS

Intrinsic obstructions are produced by congenital diaphragms, or by stenosis or atresia which follow embryonal errors of recanalization of the bowel lumen. When this occurs at one or several sites, complete congenital diaphragms from 0.5 to 4 mm. thick, diaphragms with central openings, or nonobstructive circular constrictions result. A complete discontinuity may develop, with the segments terminating as blind sacs. Complete closure and separation of the bowel lumen is referred to as aplasia, while narrowing of the lumen from a thin channel to a well-formed canal is considered stenosis. Complete closure with the ends connected by a fibrous strand is considered as atresia.

Congenital pyloric obstruction by a diaphragm is rare. Benson and Coury reported a case they believed to be the second in the literature. Partial obstruction by a perforate diaphragm is uncommon, and may go unnoticed if sufficient emptying of the stomach takes place. Perforation of the stomach in a newborn as a complication of pyloric atresia was mentioned by Burnett and Halpert. Other defects such as pyloric or duodenal atresia with gastric dilatation is predisposing to gastric perforation because of congenital muscular weakness.

Congenital obstructions of the small intestine are relatively common. Duodenal obstruction occurs approximately once in 20,000 births. Duodenal atresia appears at three basic sites. These are proximal and distal to the ampulla of Vater and at the ampula, with secondary biliary tract obstruction. The most frequent location is distal to the ampulla.

Congenital atresia and stenosis of the small intestine occurs approximately once in 6,000 births. As reported by Gross, the incidence of stenosis in 71 cases was duodenal in 39, jejunal in 5, ileal in 19, at the ileocecal valve in 6, colonic in 1 and multiple in 1. The position of atresias in 140 cases was reported as duodenal in 32, jejunal in 19, ileal in 72, at the ileocecal valve in 2, colonic in 6 and 9 were multiple. In a review of 52 cases of intestinal obstruction between the duodenum and anus in the neonatal period, Davison and Harlan noted that atresia and stenosis accounted for 24, including 4 of imperforate anus. Sixteen were due to aganglionosis of the colon. There were 4 with bowel malrotation, 2 with diaphragmatic hernia, 1 of meconium ileus and 2 cases of acute peritonitis and 1 of chronic peritonitis in this group. Prematurity was present in 8 out of 32 cases reported by Moore. Association with other congenital anomalies is frequent.

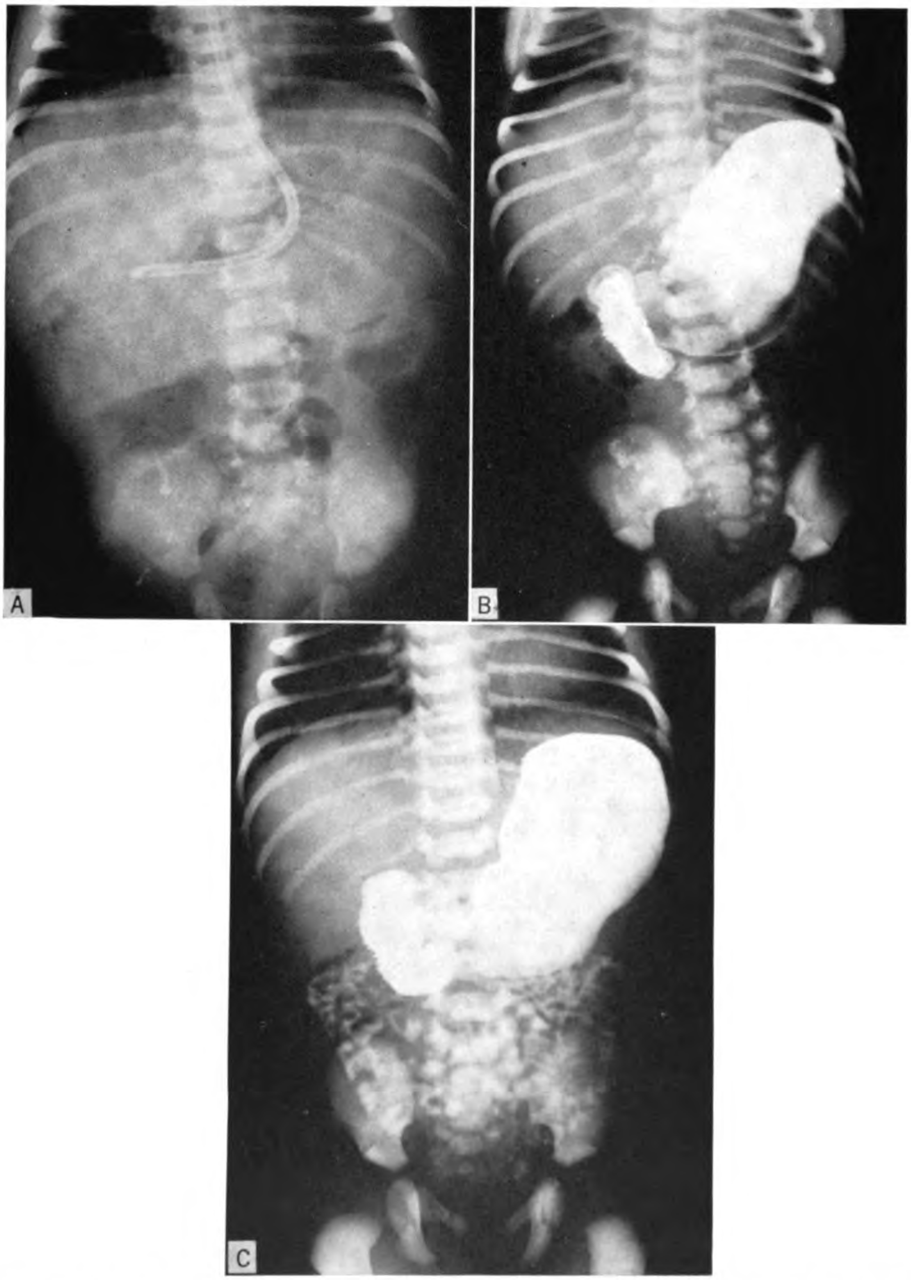

FIG. 38.—*A*, Scout film of abdomen of a 4-day-old infant with vomiting due to duodenal stenosis. The scattered loops of small intestine in the mid-abdomen are not indicative of a specific condition, although they suggest obstruction.

B, Same patient after introduction of 20 cc. of 25 per cent Hypaque. Note the abrupt termination of the descending duodenum.

C, Same patient 30 minutes later. The block at the lower duodenum is again visible, but some Hypaque has passed into the lesser bowel. At operation the root of the mesentery was found to be horizontally placed and high. The small bowel had rotated about the superior mesenteric vessels, obstructing the duodenum. The duodenal wall was thickened and the lumen stenosed in its distal portion.

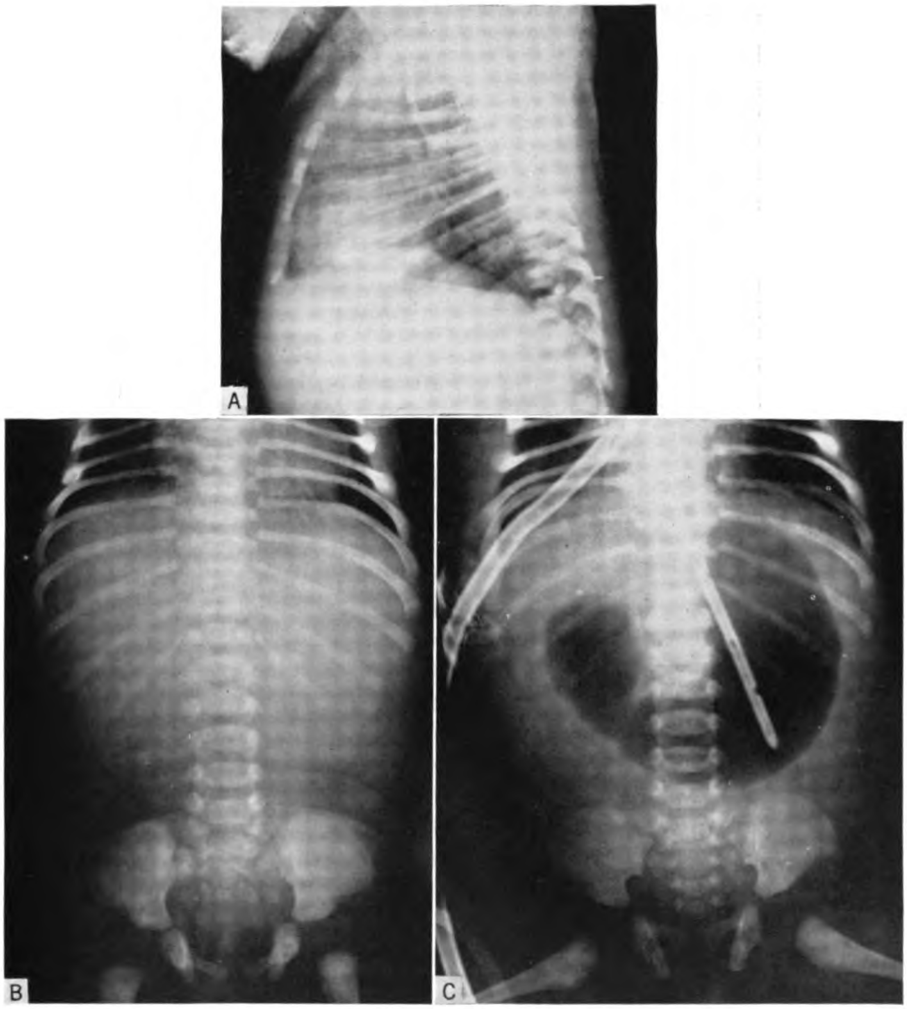

FIG. 39.—*A*, Lateral chest roentgenogram of a newborn female Mongol with difficulty in feeding due to esophageal atresia. The esophagus terminates in a blind pouch. Verified at operation.

B, Plain film of abdomen made before operation. No gas is seen in the gastrointestinal tract. This was attributed to the esophageal atresia.

C, The patient did not do well after operation. Fifty cc. of air were injected into the nasogastric tube, disclosing a dilated stomach and duodenum. Duodenal atresia was diagnosed. At necropsy this was confirmed. The patient also had an Eisenmenger deformity of the heart.

Complete gastric obstruction in the neonatal period is rare, but may be suggested by distension of the stomach in the absence of gas from the small intestine. As with the more frequently encountered duodenal obstruction, vomiting is a prominent symptom. If obstruction is above the ampulla of Vater, no bile is present in the vomitus. Bile is present in the vomitus with obstructions below the ampulla. The presence of small amounts of intestinal gas suggests the presence of stenosis. Atresias appear more frequent with proximal intrinsic duodenal obstruction, while stenosis is more common with distal obstructions. Duodenal atresia produces symptoms within the first 5 days of life. Stenosis may be asymptomatic for a much longer time. With duodenal atresia the

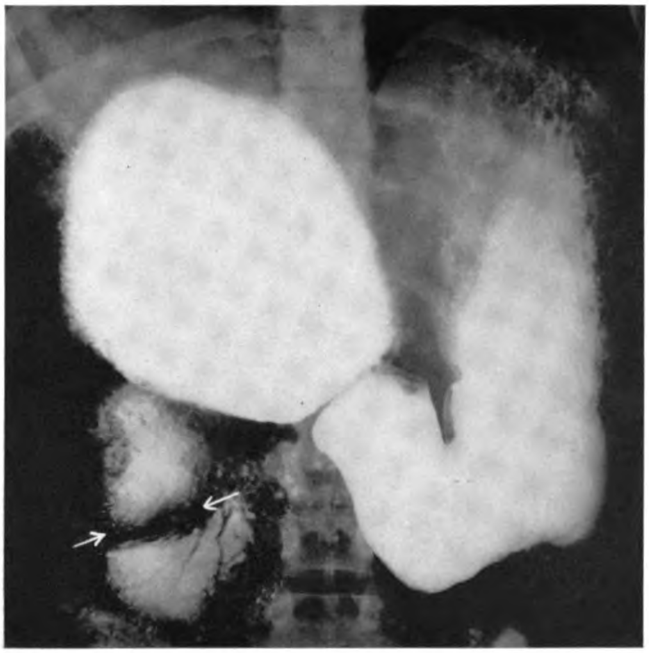

FIG. 40.—Megaduodenum in a 23-year-old girl with a congenital duodenal diaphragm and stenosis at the second and third portions, verified at operation. (Drucker V. and Cohen, E. S., courtesy of Am. J. Roentgenol., *55*, 726, 1946.)

gastric dilatation may be overshadowed by enormous duodenal distension, resulting in some cases in an hour-glass appearance with no gas distal to the obstruction. In these cases it is helpful to determine the location of the cecum, since colonic malposition may point to the presence of an extrinsic obstruction and volvulus.

Atresia of the jejunum and ileum causes obstruction. The diagnosis often is made at operation, when concomitant congenital defects frequently are found. The importance of recognizing the presence of multiple atresias is self evidenr.

Large bowel atresia is relatively uncommon. Vomiting is an outstanding symptom, and these patients are acutely ill with distended abdomens, edema of the flanks and scrotum or vulva, and anorexia. If only stenosis is present, symptoms may be minimal and of long duration. Rapidly increasing distension above the obstruction is sometimes followed by perforation of a localized area of ischemic necrosis. Differentiation of this condition from meconium obstruction often is not possible.

An interesting example of multiple atresias was seen in a 2-day-old infant who had difficulty in feeding (Fig. 39). A scout film of the abdomen showed no gas in the intestinal tract. A lateral chest film revealed air in the esophagus only up to its midportion. The diagnosis of esophageal atresia was confirmed at operation. The patient did not improve, and reëxamination of the abdomen showed only a small amount of air in the stomach. Insufflation of air into the stomach through a nasogastric tube disclosed an obstruction in the duodenum. At necropsy a congenital atresia of the duodenum was found. The patient also had an Eisenmenger malformation of the heart.

The complexity of radiologic differential diagnosis of atresias from other intestinal obstructions is exemplified by a 10-month-old girl who had cramps, difficulty in feeding and inability to gain weight. Direct examination of the abdomen showed pronounced jejunal distension, with little gas in the stomach. The duodenal loop was markedly dilated, its descending limb terminating in an arrow-shaped projection. At operation a

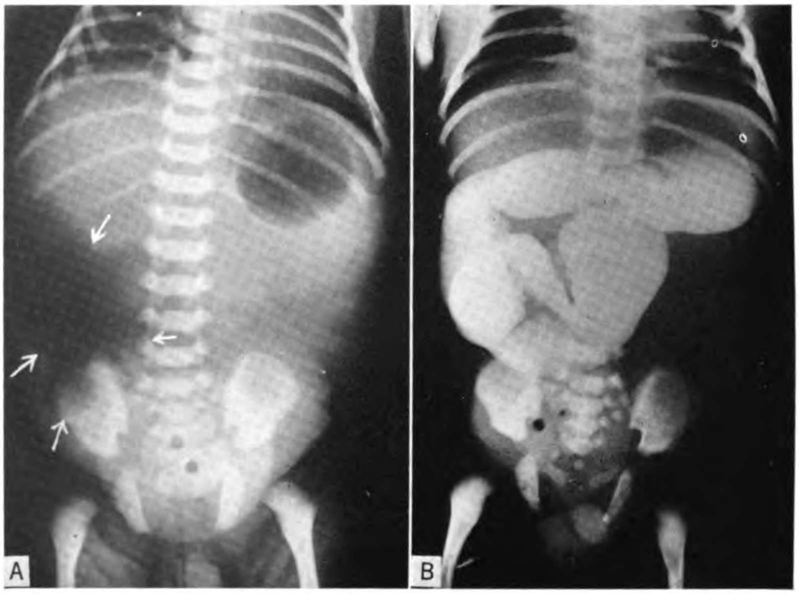

Fig. 41.—*A*, Scout film of abdomen of 3-day-old premature infant with bile-stained vomiting due to intestinal atresia. No meconium had been passed and a small mass was felt in the lower abdomen. There is a markedly distended loop of bowel in the right abdomen.

B, Same patient 10 hours after a barium meal. The lesser bowel is obstructed in the right lower quadrant. At operation the proximal jejunum was dilated and ended at about 6 inches. Fibrous bands represented the remainder of the small intestine. Two small dilated bits of bowel represented cecum and ascending colon. The remainder of the colon was reduced to cord-like strands.

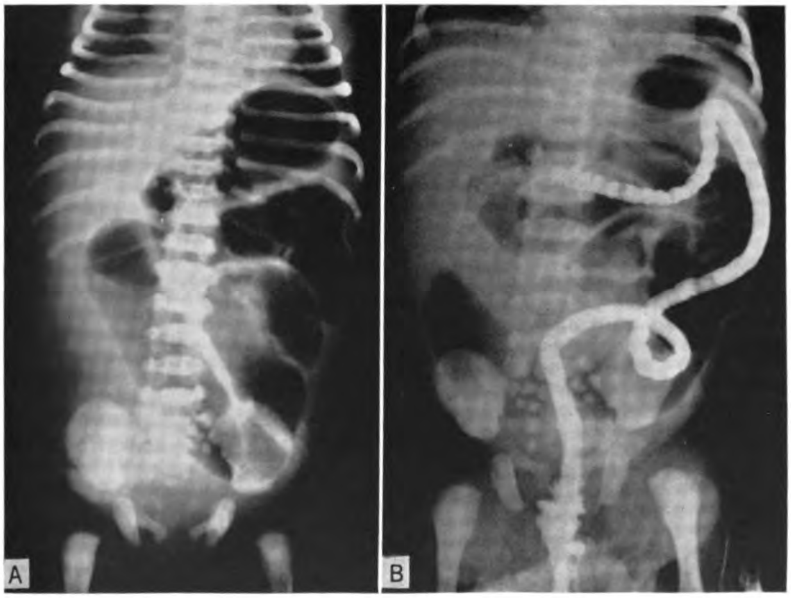

Fig. 42.—*A*, Scout abdominal film of 1-day-old infant with bilious vomiting. No meconium had been passed. The lesser bowel is markedly dilated in its jejunal portion but no gas is distal to this region.

B, Same patient after introduction of barium into the rectum. There was a block in the right upper quadrant, presumably ah igh cecum. At operation there was a congenital fibrous cord replacing the jejuno-ileal junction.

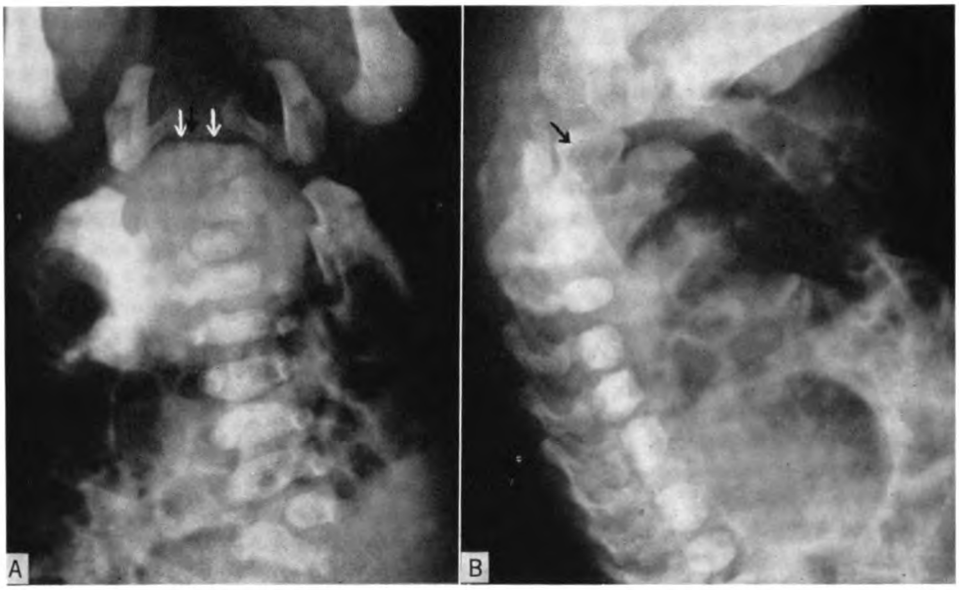

FIG. 43.—*A*, Newborn infant with imperforate rectum and rectovesical fistula. Inverted film in anteıoposterior position showing gas in bladder (arrows).

B, Same patient, lateral inverted film showing gas in imperforate rectum and in bladder (arrows). Note the congenitally malformed sacrum.

congenital diaphragm was found obstructing the midjejunum. The duodenojejunal junction was obstructed by a congenital band, and an elongated mesentery permitted a volvulus of the small bowel. In another newborn infant the coexistence of atresia at the jejuno-ileal junction and atresia of the colon was confirmed at necropsy. In this patient (Fig. 41) the stomach was normal and the duodenum dilated. The proximal jejunum was sausage-like for about 6 inches, ending abruptly in a fibrous band which represented the remainder of the small bowel. Two sausage-like pieces of bowel in the right lower quadrant represented the cecum and ascending colon. The remainder of the colon was represented by cord-like bands. This patient expired at the end of 3 days. No meconium had been passed, and the patient had vomited bile stained fluid. A catheter could be passed into the rectum for only an inch. Still another infant had ileal stenosis together with stenosis of the descending colon confirmed at necropsy. The ileum was replaced by a fibrous band, resulting in marked dilatation of the duodenum, the jejunum and upper ileum. The descending colon was only a fibrous strand, while the rectum remained patent.

ANORECTAL CONGENITAL MALFORMATIONS

Congenital obstructions at the anorectal area occurs as the result of developmental abnormalities at about the seventh or eighth week of fetal life. These occur once in about 5000 births. Four malformations are encountered. These are anorectal stenosis at or above the anus, membranous obstruction with imperforate anus, imperforate anus with the rectal pouch ending blindly above the anus, and a normal anus and rectal pouch ending blindly in the sacral hollow. Gross reported on 507 cases, 270 in males and 237 in females. The third type of deformity, imperforate anus with the rectal pouch ending blindly above, occurred in 443. Fistulas connecting the rectum with the genitourinary system or the perineum occurred in 71 per cent, more often in females. In males the fistulas were between the rectum and urethra or perineum in front of the anus. In females rectovaginal or rectoperineal fistulas were most frequent, while rectovesical and rectourethral connections were rare.

Usually the absence of a rectal opening is apparent at birth. If an opening is present atypical passage of meconium or structural malformation is readily recognized. Sometimes recognition is delayed if meconium is passed. With obstruction, the effects of block become apparent within a day or two.

Anorectal malformations are recognized radiologically by identification of gas within the bowel in proximity to the point of obstruction. When no external opening exists, the infant should be inverted so that gas may rise to the highest point in the bowel before the films are exposed. A metal marker should be placed in the anal dimple to help determine the extent of the separation between the blind end of the rectum and the perineum. It is advisable to wait about 24 hours for gas to reach the lower bowel, inasmuch as the examination may be misleading in the absence of rectal gas. Fistulous tracts may be injected with radiopaque material.

Bryson and Chartres reported a case of anorectal atresia in which a dilated small intestine simulated the appearance of the rectum. These authors remarked that a high incidence of multiple atresia exists with anorectal malformations. Dilated loops of small intestine entering the pelvis sometimes cannot be distinguished from the rectum on inverted examinations. Early vomiting and other obstructive signs may indicate the possibility of concomitant intestinal obstruction due to other lesions. Other malformations also are rather common, as seen in 3 of our patients with imperforate rectums who also had rectovesical fistulas together with malformations of the vertebral column.

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CYSTIC FIBROSIS OF THE PANCREAS

Cystic fibrosis of the pancreas is a hereditary disease of children which involves the mucous glands throughout the body as well as the pancreas. With accumulation of a substance believed to be abnormal mucous, dilatation of the various secretory glands takes place. This affects the pancreas, the liver, the lungs and the small intestinal glands. Markedly increased excretion of electrolytes takes place when the sweat glands are disturbed, and the parotid secretions likewise are abnormal.

In infancy this disease impairs the function of the pancreas and lungs. A diffuse, variable bronchopneumonia is predicated upon bronchial obstruction by mucous plugs. Generalized obstructive emphysema, chronic bronchopneumonia and secondary infections are superimposed. Acute respiratory distress may be produced by blockage of a major bronchus. Progressive obstruction and repeated infections result in bronchiectasis, chronic atelectasis, abscess formation, pulmonary insufficiency and ultimately death.

Pancreatic involvement begins in intrauterine life, resulting in gradually progressive fibrosis of connective tissue with atrophy and disappearance of the parenchymal tissue. In some a considerable inflammatory reaction is present. The islets of Langerhans remain normal. As ductal obstruction and retention of secretions progress, the pancreas becomes cystic, with normal islets scattered in the fibrotic parenchyma. Destruction of secreting tissue results in absence of pancreatic enzymes, and as a result large amounts of fat and nitrogen are excreted in the stools, which become bulky, greasy and foul. The digestion of carbohydrates is less affected. Malnutrition develops because the patient is unable to utilize the ingested food.

In from 10 to 15 per cent of infants with cystic fibrosis, pancreatic achylia is associated with abnormally thick tenacious mucous which can produce the obstructive condition known as meconium ileus. Extensive distension and rotation of the bowel may simulate volvulus. The colon distal to the obstruction remains small, as easily demonstrated by contrast enema. Meconium is an irritative substance, and if any enters the peritoneal cavity through a rupture of the bowel, meconium peritonitis ensues. Perforation of the intestinal wall occurs before, during or after birth. There are no clinical nor radiologic criteria for diagnosis other

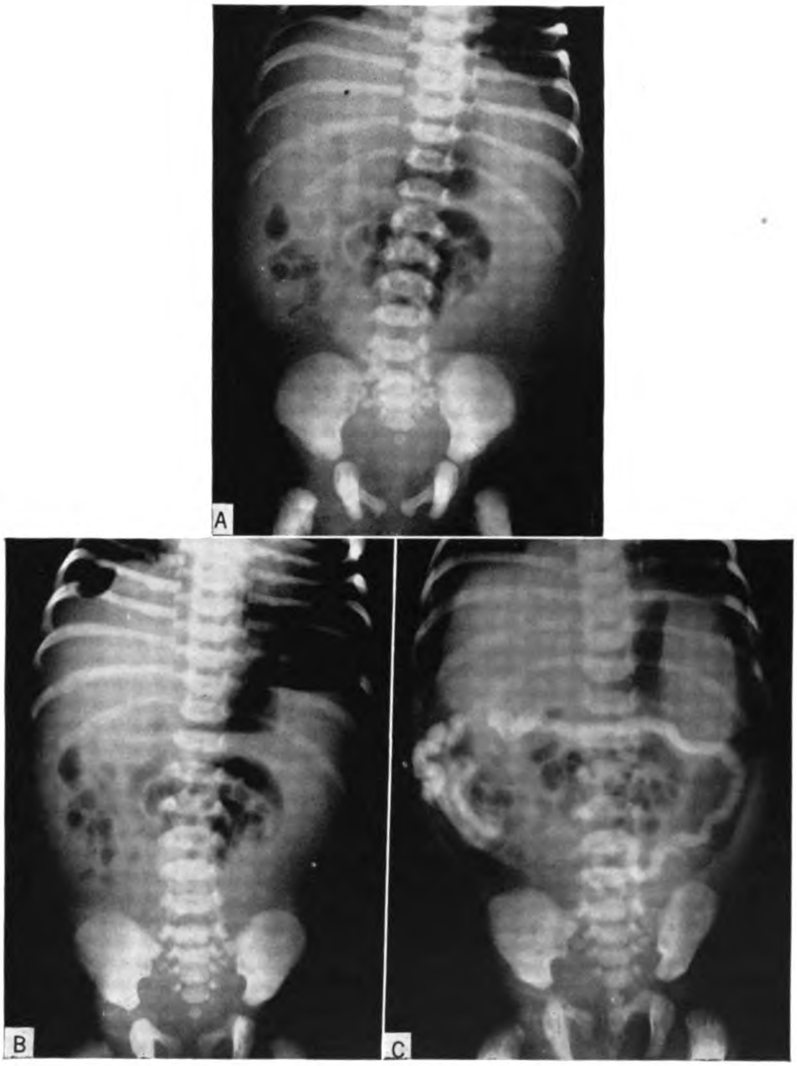

FIG. 44.—*A*, Meconium peritonitis in a newborn infant due to rupture of ileum just above an area of congenital atresia with hypertrophy of the proximal bowel. The intestinal loops in the midabdomen are distended but no specific change is present.

B, Same patient, erect film reveals pneumoperitoneum with gas beneath the right and left diaphragm. This may account for the relatively mild intestinal dilatation.

C, Same patient after introduction of barium into colon revealing undilated bowel with high cecum. The extent of the pneumoperitoneum has increased. Diagnosis verified at operation.

than those of intestinal obstruction with pneumoperitoneum in many such cases.

Meconium normally reaches the ileocecal junction by the fourth month of intra-uterine life, and is in the rectum by the fifth. Anything obstructing its passage predisposes to meconium obstruction and perforation. Since fetal meconium is sterile, intra-uterine perforations sometimes heals spontaneously. In such cases intraperitoneal calcifications are sometimes present. At approximately the third day of life the meconium becomes contaminated, producing an infective peritonitis on rupture of the bowel. Occasionally

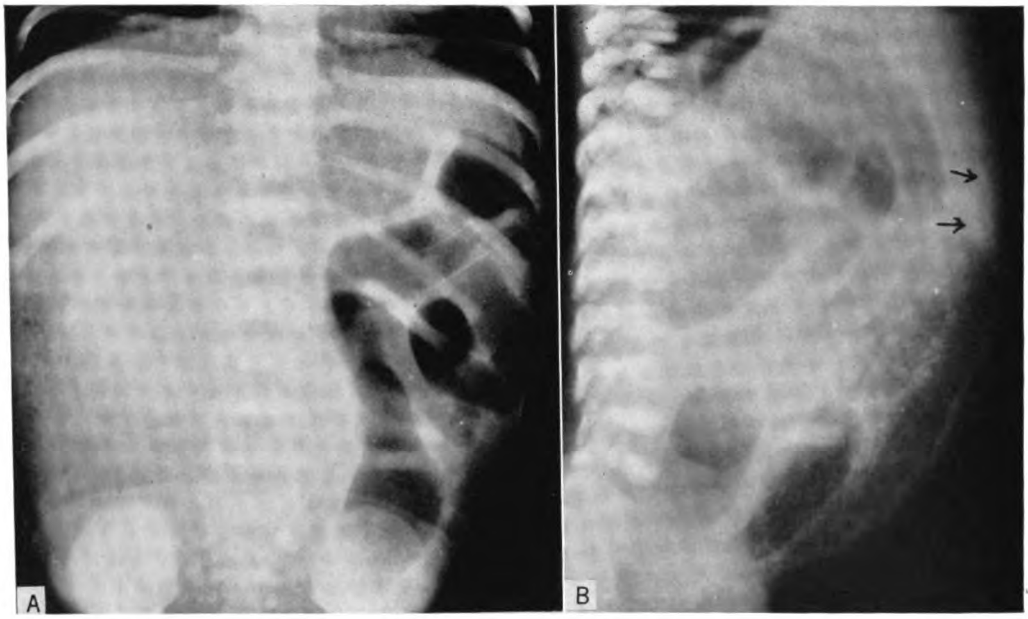

FIG. 45.—*A*, Meconium peritonitis due to perforation of ileum above atretic terminal ileum. Anteroposterior scout film reveals mottled gas and fluid shadows in right side of abdomen with dilated small bowel to the left of the midline.

B, Same patient, lateral view. Very faint linear calcifications are present along the anterior abdominal wall (arrows).

an intrapartum peritonitis is identified by distension at birth in a distressed infant. Pneumoperitoneum may appear early and should be sought for on erect roentgenograms.

In meconium peritonitis the calcifications form along the surfaces outside the intestinal tract. Another helpful sign is the presence of small minute bubbles of gas mixed with meconium in the distal small bowel. The dilated small intestinal loops show no abrupt termination of gas to indicate the site of obstruction. As a rule gas is absent from the colon. Erect roentgenograms disclose fluid levels in no way different from those of other obstructive lesions. Intra-abdominal calcifications also appear within the intestinal tract, as for example within the lumen of atretic small intestinal loops.

Meconium peritonitis may exist secondary to perforation of the fetal intestine without obstruction. Franklin and Hosford reported 2 such patients, both recovering after operation, where the intestine was found imbedded in a thick fibrous layer. In 1 case there was marked calcification of the parietal and visceral peritoneum at birth. While bowel rupture is often associated with obstruction, perforation also occurs in its absence. A case with intra-abdominal calcification indicative of meconium peritonitis recognized 2 days before delivery was reported by Harris, Bemuth and Harris. Necropsy showed no obstruction of the gastrointestinal tract, nor was there any appreciable amount of meconium within the bowel. Nash and Smith mentioned that meconium peritonitis can result from leakage through congenital defects of the bowel wall, as in patent Meckel's diverticulum. Occasionally meconium impacted in the colon may cause pneumoperitoneum due to rupture of the bowel proximal to the obstructive site. Intestinal atresia in the newborn also may produce pneumoperitoneum with passage of meconium into the peritoneal cavity. This is accompanied by extreme distress, with distension, cyanosis, vomiting, and edema of the flanks, scrotum or vulva. A contrast enema which reveals an undilated bowel

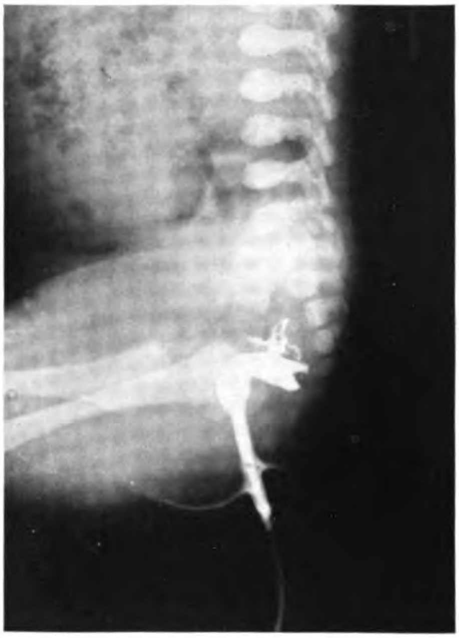

FIG. 46.—Low colonic obstruction from meconium plug in a newborn infant who did not have meconium ileus.

may help identify an obstruction higher in the intestines.

Cystic fibrosis results in localized foci of biliary fibrosis and obstruction leading to cirrhosis. Usually this causes no symptoms. In a few cases hepatosplenomegaly, hypersplenism, gastrointestinal bleeding or ascites have caused serious complications. A review by diSant'Agnese delineates the general aspects of diffuse cystic fibrosis and the interrelationships between pulmonary and gastrointestinal disorders, involvement of the exocrine glands, the liver and the appearance of diabetes mellitus.

Occasionally newborn infants are unable to expel a residual plug of meconium either because of its altered consistency or impaired mobility. Clatworthy, Howard and Lloyd reported 9 such cases in infants from 1 to 6 days old, 8 of which were males. In none had normal meconium been passed. Abdominal roentgenograms disclosed a gaseous pattern consistent with low intestinal obstruction. Digital examination and enemas yielded meconium with subsequent relief of symptoms. None developed pancreatic fibrosis or congenital megacolon. These authors noted that differentiation of meconium plugs and bowel dilatation from true Hirschsprung's disease may be difficult.

Later, achylia due to cystic fibrosis of the pancreas is characterized by foul large fatty stools, increased nitrogen excretion in the stool, and the effects of poor vitamin A absorption. Malnutrition follows, with retardation of growth and development. Long-standing pulmonary infections are frequently present. A second smaller group has pancreatic insufficiency without pulmonary disease. These may be mistaken for idiopathic celiac disease patients. A still smaller group has chronic pulmonary disease without intestinal manifestations. The fourth, smallest group are those with cirrhosis of the liver and portal hypertension. In these gastrointestinal bleeding may be the chief complaint, with neither gastrointestinal distress nor pulmonary symptoms. An interesting change occurs with marked dehydration because of excessive chloride excretion during hot weather. The determination of chlorides in the sweat sometimes is of considerable diagnostic significance. Demonstration of absence of pancreatic enzymes on duodenal assay is helpful, even though this is not absolutely diagnostic inasmuch as it may not be centered when pancreatic involvement is only mild.

Gastrointestinal radiographic examination in children surviving into their second year of life and longer often reveals segmental dilatation of the lesser bowel, with flocculation and clumping of barium and slow motility. This "deficiency" pattern, while not specific, is of diagnostic help in some cases.

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MYENTERIC GANGLIONIC DEFECTS

Aganglionic Gastrointestinal Conditions.—Congenital malformations of the gastrointestinal myenteric plexuses may extend from the distal esophagus to the anus. Manifestations of such dysfunction occurs from birth to old age, the best known form being that affecting the colon and designated as Hirschsprung's disease. The basic defect is considered to be a failure of development or maturation of the ganglion cells of the myenteric plexus of Auerbach rather than a degenerative process. These plexuses, situated between the two muscular coats of the bowel, may be completely absent with no ganglion cells, or cells may be present in reduced numbers. Small nerve trunks may be identified. The bowel wall in the aganglionic segments appears normal, the lesion being identified only on histologic examination. With certain deficiencies of the myenteric plexuses hypertrophy of the adjacent bowel is encountered.

Deficiencies in the myenteric plexus at the cardio-esophageal junction is associated with *achalasia of the esophagus*. This occurs in two forms. The first, present in about 75 per cent of cases, is pronounced esophageal dilatation with marked relaxation and redundancy of its walls. The terminal few centimeters of esophagus are identified radiologically by an occasional trickle of barium into the stomach. At operation the terminal esophagus is slender, with abnormally thin musculature, while the esophagus above is enormously dilated with thickened walls. The second type of achalasia presents less dilatation, with active, sometimes irregular peristalsis and considerable retroperistalsis. In this group the lower esophagus as seen at operation is thickened, with markedly hypertrophied circular muscular fibers. Sweet believes that there is no evidence that one type evolves from the other.

Evidence has appeared recently that *hypertrophic pyloric stenosis* is an aganglionic disorder. This condition is familial, occurring frequently in first-born males. Initial symptoms appear at from 2 to 6 weeks, and the clinical picture of projectile vomiting associated with an epigastric mass is well known. The lesion is a firm, hypertrophied, almost cartilaginous muscle with variable obstruction. While the normal infant pylorus is from 1.0 to 1.5 cm. long, a hypertrophied pylorus measures up to 3 cm. in length. The width of a normal infant pylorus reached 0.3 cm., while a hypertrophied pylorus attains 0.7 cm. Microscopic examination shows thickening of the submucosa by edema and hypertrophy of the circular muscle with no concomitant increase in the thickness of the longitudinal muscle or its connective tissue. Recent studies by Belding and Kernohan of the myenteric plexus and pyloric muscle in hypertrophic pyloric stenosis dis-

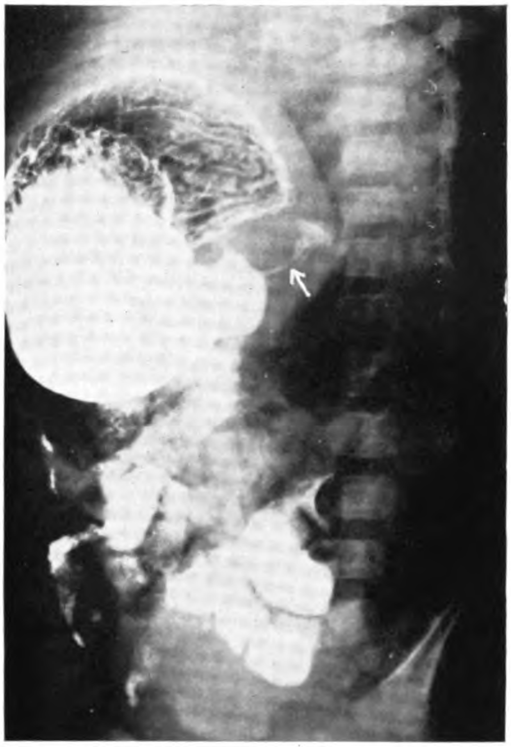


FIG. 47.—Four-week-old male infant with hypertrophic pyloric stenosis. A classical string sign is present (arrow).

closed a definite decrease in the number of ganglion cells and nerve fibers, with no significant deviation from normal above or below the pylorus. Friesen, Boley and Miller attributed histologic abnormalities of the ganglion cells to failure of development or maturation rather than degeneration.

Radiographic examinations for hypertrophic pyloric stenosis in infancy usually is reserved for doubtful cases. A review of our material for the past 15 years was made. Gastric dilatation usually is inconstant, and may be absent if the examination is made soon after a bout of vomiting. We feed the infant the contrast medium using a bottle, or if this is too time consuming, a small nasogastric catheter is passed and from 30 to 60 cc. of opaque material is injected under fluoroscopic control. If the diagnosis is soon evident by demonstration of a classical "string sign," spot films are made and the examination discontinued after the residual contrast material is aspirated. If water soluble contrast substances are used removal of the gastric content is unnecessary. If a string sign is not quickly apparent, in order to keep radiographic exposure to a minimum, it is advisable to wait about 20 minutes before proceeding with the examination.

The configuration of the antrum is quite variable. In some the string sign is of a tapering variety, while in others it is practically at right angles to the antrum. The rate of gastric emptying is inconstant. In some sufficient barium enters the intestines within an hour to cause some doubt as to the diagnosis.

In 3 recent patients the string sign was transient and at times absent. No significant gastric emptying delay was noted. However symptoms persisted and reexamination about 2 weeks later disclosed a classical string sign. The diagnosis thereupon was confirmed at operation. In 1 case the tip of the catheter passed through the pylorus into the duodenum. This raised the question as to whether or not the pyloric opening is fixed. It is my impression that variability in the degree of opening of a hypertrophied pylorus does occur. Demonstration of a well-formed duodenal bulb with hypertrophic pyloric stenosis is uncommon, possibly due to an insufficient amount of contrast medium entering the duodenum to distended it. Good formation of the bulb and sweep were observed when a relatively free passage of barium was seen.

Infants with hypertrophic pyloric stenosis often have little or no gas in the lesser bowel, attributed to mechanical effects of pyloric obstruction. However, Feinberg, Margulis and Nice pointed out that the absence of intestinal gas was due to neuromuscular dysfunction caused by dehydration rather than to mechanical obstruction. They reported that dehydrated infants showed relatively little intestinal gas, while roentgenograms made after correction of dehydration showed a striking increase in the gaseous content of the bowel. Projectile vomiting like that of hypertrophic pyloric stenosis has been observed with adrenal cortical insufficiency by Weens and Golden. They presented a case with persistent narrowing of the pyloric area, designating this as "pseudopyloric stenosis."

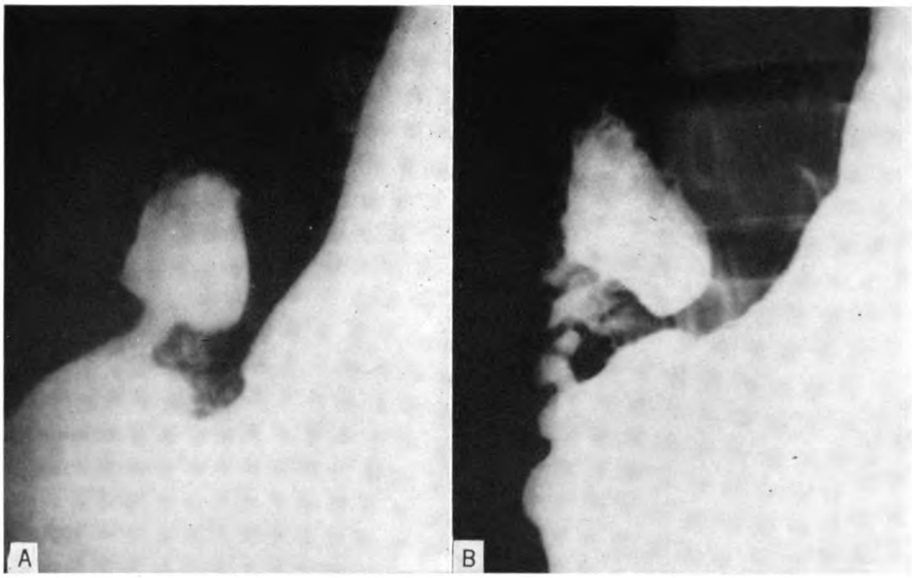


FIG. 48.—*A*, Benign hypertrophic pyloric stenosis in a 62-year-old woman, verified at operation. *B*, Another case of benign hypertrophic pyloric stenosis with marked antral irritability in a 61-year-old woman.

The possibility of reflux esophagitis with shortening of the esophagus in infants who vomit excessively must be kept in mind. Astley and Carré mentioned 5 children with hypertrophic pyloric stenosis who also had partial intrathoracic stomachs.

In considering gastric obstructions, other congenital lesions must be excluded. Touroff and Sussman reported a congenital membrane causing obstruction in a premature infant. Partial pyloric obstruction due to mucosal diaphragms have been observed in adults also. Such cases were reported by Rota and by Swartz and Shepard. Functional obstructions with no anatomic cause sometimes occur with intracranial lesions. Evans mentioned a case which at necropsy disclosed a hemorrhagic ulcerating gastritis and duodenitis.

The continuance of hypertrophic pyloric stenosis into adult life is not rare. A persistence of the infantile form may first become apparent at from 30 to 60 years of age. North and Johnson gathered 59 such cases from the literature, and added 5 more. An additional 4 cases were recently published by Kleitsch. The clinical picture is one of intermittent gastrointestinal disturbances with epigastric pain and vomiting, sometimes relieved by food. Concomitant gastric ulcerations have been observed relatively often. Radiologically, these lesions simulate annular pyloric cancers, and the question often cannot be answered without surgery. We observed 6 patients from 38 to 60 years old with this condition. One, a 60-year-old male, had had epigastric pain and heartburn for at least 15 years. Gastrointestinal examination was indicative of an annular pyloric tumor, and operation was recommended. However, enthusiasm for surgery disappeared when the patient returned with a sheaf of films made during the preceding 11 years showing the condition unchanged. A small ulcer was present at the base of the bulb, which probably accounted for the patient's symptoms. In all 6 patients the duodenal bulb and sweep were well outlined. The pyloric canal did not present an elongated string sign, but varied in diameter from 1 to 1.5 cm. and in length from 1.5 to 2.5 cm. Gastric retention varying from approximately 25 to 75 per cent at 6 hours was present in all.

One adult patient of unusual interest had a marked deformity of the antrum on successive studies, believed to be either a hypertrophied pyloric muscle or an annular carcinoma. At operation, to our surprise, no deformity was present. I still recall the disdainful look of the surgeon as he picked up the pylorus and announced that it was obviously normal. The first assistant thereupon picked up the pylorus with a mouth-tooth forceps, whereupon it immediately contracted upon itself and assumed a configuration like that seen on the roentgenograms. The muscle became almost stony hard, and the overlying serosa blanched markedly. When the forceps were removed the stomach relaxed and gradually resumed its normal appearance. When the surgeon repeated the maneuver, the antrum again went into spasm and relaxed when pressure was removed.

The fate of patients operated upon for hypertrophic stenosis was reported recently by Nielson and Roelsgaard. They reviewed 45 adults who had had pyloric stenosis treated medically. In 35 there were characteristic changes in the prepyloric area. In addition, peptic ulcers were present in 14. The pyloric defect described was a permanent narrowing with an abrupt transition to the adjoining normal antrum. Armitage and Rhund noted that pyloric hypertrophy persisted into adult life when treatment had been gastroenterostomy instead of pyloromyotomy during infancy. The surgical treatment of hypertrophic pyloric stenosis in infancy was summarized by Gross's statement "over 99 per cent of babies with pyloric stenosis (even when accompanied by other abnormalities or by prematurity) have survived operation and have been completely and promptly relieved of their symptoms." The controversy as to whether medical or surgical treatment is preferable still exists.

Aganglionic Defects in the Small Intestine.—Absence of ganglion cells in the myenteric plexus of the duodenum occurs infrequently. Thirty-five such cases were collected by Barnett and Wall. These were considered related to ganglionic deficiences in the colon and ureters. A high familial incidence was present. The usual complaints were epigastric pain, nausea, vomiting, tenderness, and occasionally a mass was felt in the epigastrium. These authors reported a 30-year-old woman who had a sudden massive hematemesis. Gastrointestinal examination disclosed a grossly dilated and hypertrophied duodenum, confirmed at operation. No evidence of mechanical obstruction was found.

Involvement of the ganglion cells of the jejunum and ileum in an isolated form is rare. I have been unable to find such cases in the available literature. However, there are cases in which small intestinal aganglionosis occurs as part of a generalized picture. Wangensteen mentioned that he had seen 1 patient in whom the entire gastrointestinal tract was aganglionic. Zuelzer and Wilson and others have noted the presence of ileal aganglionosis in association with colonic involvement.

Megacolon.—Marked dilatation of the colon falls into three generalized groups. Aganglionic defects associated with marked dilatation of the bowel is the true Hirschprung's disease. First recognition of the neurogenic nature of the disorder was by DallaValle and by Cameron. Current interest stems from the work of Zuelzer and Wilson, Whitehouse and Kernohan, and the contributions of Swenson, Neuhauser and their associates are fundamental in today's management of the condition.

Megacolon in the absence of aganglionic defects occurs as a functional disorder in which fecal impaction is caused by deficiencies at the anal sphincter producing an insidious obstruction. Pronounced dilatation of the bowel also is associated with elongation and poor muscular bowel tone of no specific origin, with diverse factors such as a spastic sphincter, faulty bowel habits, or excessive water absorption from an unusually elongated sigmoid. An acquired form of megacolon prevalent in Brazil was reported by Raia in adults, supposedly associated with hypovitaminosis B_1. Microscopic examination of the colon in these patients disclosed histologic changes comparable to those seen in children with Hirschprung's disease, with all stages of ganglion cell fragmentation. Dilatation of the bowel resulted from fecal stasis due to asynchronism between the peristalsis of the descending colon and the terminal segments of sigmoid and rectum.

Congenital aganglionic megacolon sometimes is apparent at birth. The length of the

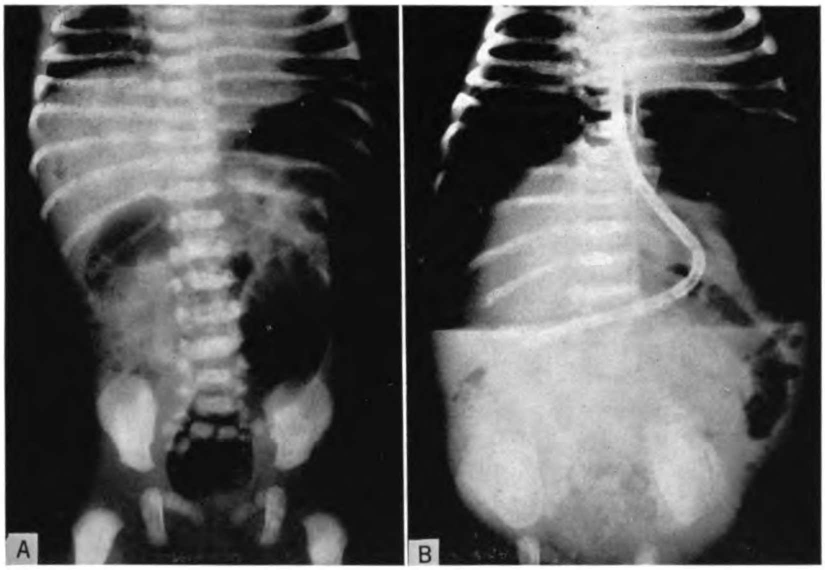


FIG. 49.—*A*, Infantile megacolon due to aganglionosis of the rectum. Note the dilatation of the sigmoid. No obstruction was found on digital examination and the diagnosis was not made.

B, The same patient 1 week later. An advanced pneumoperitoneum is present. At necropsy there was "neurogenic dilatation of the sigmoid with obstruction, perforation and pneumoperitoneum." A review of the slides disclosed aganglionosis of the rectum.

involved segment varies from a small patch at the anus and lower rectum to one which includes the entire colon and the terminal ileum. The most common area of involvement is a relatively short portion of rectosigmoid, with constriction of the involved segment. The bowel distal to this also contains abnormal ganglion cells. A physiological obstruction is produced, resulting in marked dilatation of the bowel above the constricted area. The extent of the aganglionic area and of the constricted area are not necessarily identical, the bowel beneath possessing variable potential for dilatation. Incidences of skip areas of aganglionosis in the bowel have been reported. Limitation of aganglionic defects to the hepatic and splenic flexures also have been observed. Ragins and Prohaska described megarectum due to aganglionosis of the anorectal area in an 11-year-old male born with an imperforate anus. At 12 hours after birth a high rectum was found at laporatomy after an unsuccessful attempt to reach the rectal pouch through the sphincteric dimple. This was recurrent after several colostomy openings had been established. The presence of megarectum at birth together with anorectal aganglionosis supports the idea that the aganglionosis rather than the imperforate anus was responsible for the huge rectal dilatation.

Out of 160 cases reviewed by Bodian, Carter and Ward, 13 were of the long segment variety, including 9 with involvement from anus to splenic flexure, 1 from the anus to the hepatic flexure, 1 from the anus to the ileocecal valve and 2 from the anus to the distal ileum. They also reported a 4-day-old boy with small bowel obstruction in whom the colon appeared normal. A transverse colostomy was done, but the child expired at 10 days without passage of meconium. Aganglionosis from the duodenojejunal junction to the anus was discovered at postmortem examination. Swenson reported 2 cases of Hirschsprung's disease with aganglionosis of the entire colon. Ileostomy was performed and several months later the colon was resected and ileoproctostomy accomplished. This was again emphasized by Close and Clausen, who commented on neonatal obstruction caused by myenteric agenesis of the entire colon, with the bowel within normal radiologic limits. If agan-

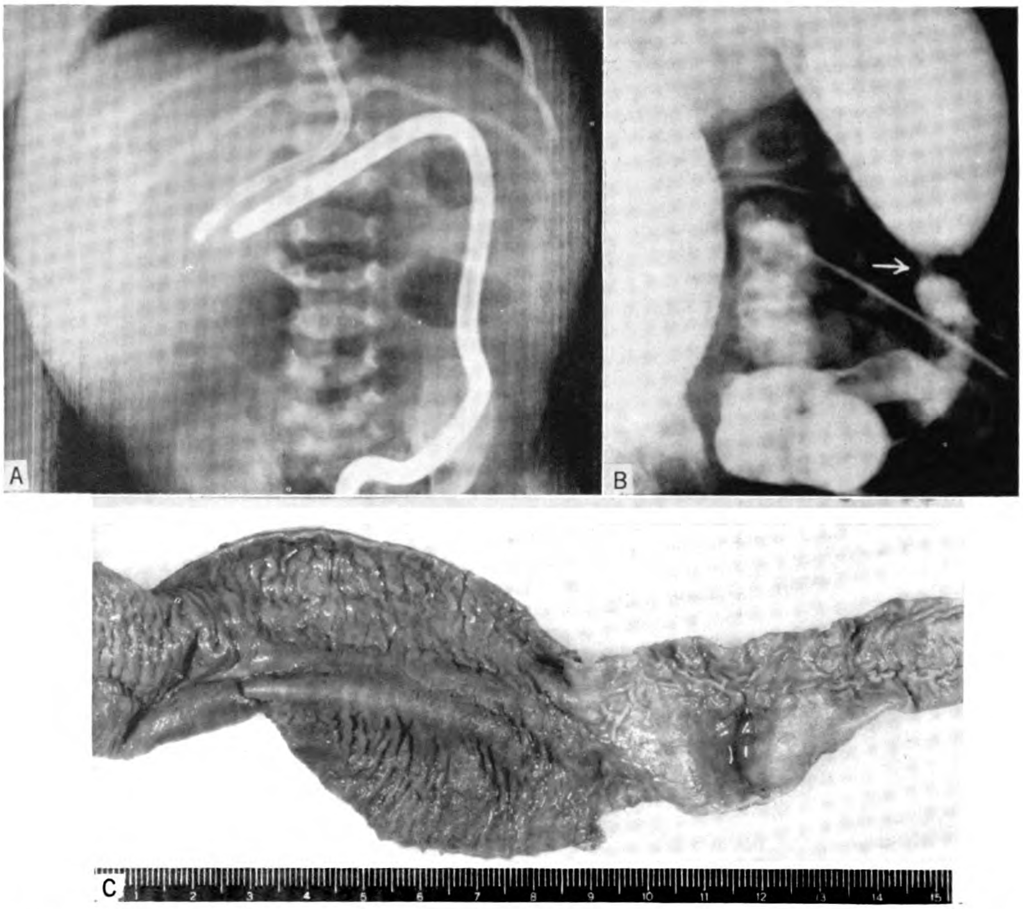


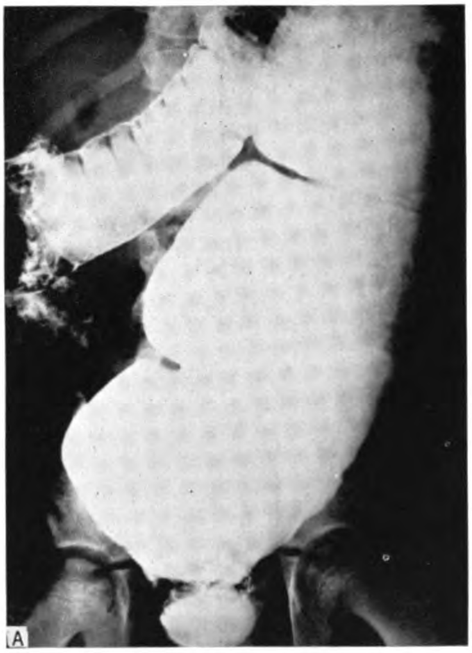
FIG. 50.—*A*, Megacolon in a 12-day-old infant with a history of vomiting, diarrhea, distention and weight loss. The vomiting became feculent. Spot roentgenogram made after a rectal tube had been threaded into the transverse colon, passing beyond an area of narrowing in the sigmoid.

B, Same patient, after instillation of Hypaque into the rectal tube while it was being withdrawn. Note area of narrowing in the distal descending colon. This was considered an area of constriction incident to aganglionosis.

C, Diagnosis of Hirschsprung's disease varified at necropsy. The bowel proximal to the aganglionic area is dilated and hypertrophied.

glionosis affects the terminal ileum, the small intestine becomes dilated, with a clinical picture of distal small bowel obstruction. This must be relieved by an ileostomy. Megcolon does not develop in these children, and the diagnosis must be suspected on clinical grounds.

The symptoms of Hirschsprung's disease are diverse. In infancy it may appear first as persistent diarrhea, or with infrequent delayed evacuations, abdominal distension and vomiting. Particularly in infancy, the colon is not grossly dilated. Small amounts of meconium and later stool may be passed, but usually bowel function is abnormal. In infants, gaseous distension is evident on scout films. As a rule, gas is lacking in the rectal ampulla. Barium enema examination is required to exclude obstruction or congenital malposition of the colon. Even though the colon is not demonstrably altered in infants with aganglionosis, retention of barium 24 yours after enema examination is suggestive of this condition.



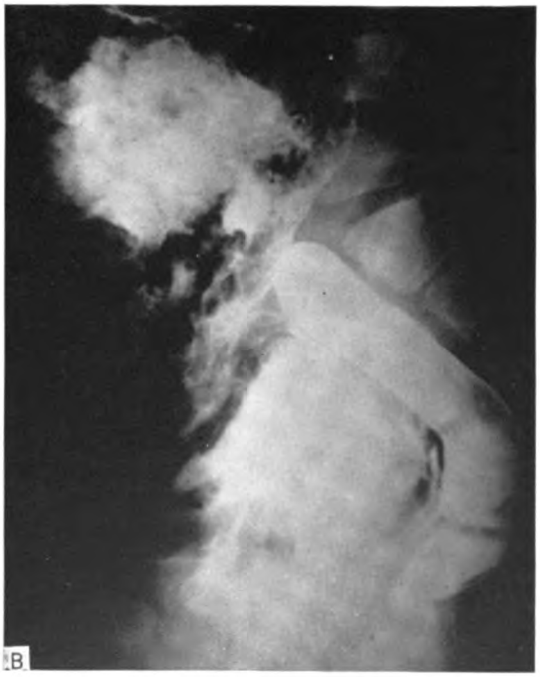


FIG. 51.—*A*, Megacolon in a 14-year-old boy with marked dilatation of the sigmoid. *B*, Same patient, lateral view. Note the narrowing of the rectum up to the rectosigmoid (arrows).

Classically, Hirschsprung's disease is diagnosed by demonstration of a relative narrowing in the sigmoid, with dilatation of the proximal colon. This can be seen on barium enema study, when properly positioned films disclose an area of narrowing of the bowel. However, when such examinations are inconclusive, it sometimes is necessary to obtain histologic evidence. Swenson, Fisher and MacMahon advise rectal biopsy, particularly when the affected area is so low that a narrow segment of bowel cannot be demonstrated radiologically. This may occur with atypical symptoms of diarrhea, intermittent intestinal obstruction and abdominal pain. Histologic confirmation is essential in planning an operation, study of resected portions of bowel during surgery being perhaps the most reliable method of determining just how much bowel must be removed.

It is often possible to demonstrate an aganglionic segment of bowel early. Berman noted that in 15 of his 19 patients examined with barium enema in the first month of life the lesion was identified. Of 7 colon examinations in the first week, 5 were diagnostic. The plain roentgenograms in 18 patients showed significant colonic distension, 12 having prominent fluid levels. The defect usually is more apparent in older children. When symptoms are mild and medical care is not sought until adolescence or adult life, the abdominal cavity becomes practically filled by a tremendous colon full of gas and stool, rendering the diagnosis quite obvious. The narrowed area in the sigmoid, and a relatively undilated rectum often are recognized.

Care must be exercised in administering a barium enema to avoid water intoxication. The enema should be prepared with normal saline instead of water, and the smallest quantity possible should be introduced. A case in which death followed a barium enema in a child with Hirschsprung's disease was reported by Jolleys. Precautionary measures were advised by Hiatt and by Steinbach, Rosenberg, Grossman and Nelson.

Infrequently aganglionic megacolon is associated with neurogenic disturbances of the bladder and megaloureter as part of disturbed parasympathetic function. Swenson postulated three general groups of this disorder, including megacolon alone, megacolon with defects in the terminal colon and bladder, and patients with intact colons but defects in bladder function alone, including some cases of megaloureter. Careful investigation may disclose minor deficiencies of bladder capacity and emptying in a fair proportion of cases of megacolon. Bladder abnormalities are relatively frequent when megaloureter is present. Potts, Boggs and White noted that in about half their cases their was diminution of ganglion cells in the bladder near the ureteral orifices with associated urinary changes in the bladder of atony, increased capacity and megaloureter.

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GASTRIC RUPTURE IN THE NEWBORN

Spontaneous rupture of the stomach is an infrequent abdominal catastrophe, usually seen during the first week of life. The symptoms are labored respiration and cyanosis, abdominal distension and tympany with no obstructive signs. The child refuses its feedings, and fresh blood may appear in the vomitus. MacGillivray, Stewart and MacFarlane noted that pressure exerted on the enlarged, gas-filled scrotum would empty the sac with a gurgling sound, only to refill when released. Prominent subcutaneous emphysema also was noted by them.

The underlying cause of pneumoperitoneum in these infants is congenital weakness of the stomach wall, usually the upper anterior aspect of the greater curvature, with all the coats involved. Sepsis with mural abscess, diffuse gastritis with necrosis, or trauma by a nasogastric tube also may cause gastric perforation, or perforation of a congenital diverticulum.

The concomitance of multiple congenital anomalies of the urinary tract with rupture of the stomach due to congenital muscular weakness was mentioned by McGillivary, Stewart and MacFarlane.

Radiologically the diagnosis is suggested by the presence of pneumoperitoneum. Inasmuch as this in itself indicates a surgical emergency, time usually is not taken for contrast examinations. It is sufficient to recall that the three causes for acute perforation of the viscus in the newborn are acute ulceration, usually duodenal, congenital pyloric or small intestinal atresia with gastric dilatation and congenital weakness of its walls, or congenital muscle defects alone.

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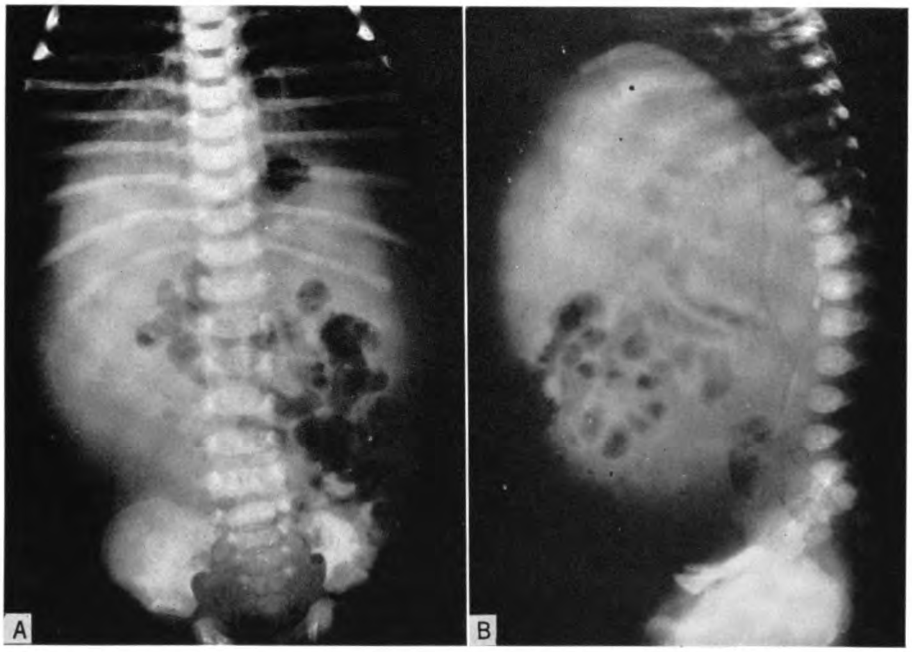

FIG. 52.—*A*, Chylous ascites in a 1-month-old male infant. The abdomen is distended with fluid and dilated loops of bowel are clustered in the midabdomen. At operation there was malrotation of the bowel with the cecum on the left side. Large lymph nodes were present in the root of the mesentery, believed to be of sufficient size to cause lymphatic block.

B, Same patient, lateral view showing mildly dilated intestinal loops.

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CHYLOUS ASCITES

Infrequently an infant's abdomen is so distended with chylous fluid that respiration is disturbed and venous return is impaired. Such accumulations may be associated with hernial sacs filled with fluid, sometimes the first hint of the process. This fluid occasionally is irritative, leading to peritoneal inflammation. Chylous ascites in infants may be due to a congenital lymphatic defect with blockage of the main trunks, sometimes accompanied by chylous effusion in the thorax and lymphedema. While the condition often is relatively asymptomatic it may occur in an acute form. Gribetz and Kanof reviewed 25 cases in children 15 years of age or less, and of these 5 had signs and symptoms of an acute abdominal disorder.

It is not always possible to distinguish between congenital causes and other lesions producing lymphatic obstruction. Kuykendall and Dederer, for instance, reported a 10-day-old infant with free peritoneal fluid, intestinal distension and progressive scrotal enlargement in whom chylous ascites was ascribed to mesenteric lymphadenitis. Lymph node enlargement due to tuberculosis, Hodgkin's disease, neoplasm or filariasis can produce a similar disorder. Direct trauma to the abdomen or thorax likewise may cause chylous effusion. In adults the condition is more likely due to a specific obstruction to lymphatic flow.

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MICROCOLON

As a rule the small colon seen with ileal meconium obstruction represents lack of dilatation because of failure of intestinal contents to enter the large intestine. This is not a congenital defect. However, rare cases of true congenital microcolon have been described. Storey and Sharp reported a newborn infant with an abnormally small colon from rectum to cecum. Its walls were intact, but the lumen was microscopic in caliber. A case of congenital microcolon with extensive small bowel and bladder malformations was reported by Lavoie. No gross or microscopic changes were present in the pancreas. Theories as to etiology vary from autonomic innervation disturbances causing achalasia to vascular abnormalities or defects of recanalization during embryonic life.

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CONGENITAL ANOMALIES OF THE GALLBLADDER AND BILE DUCTS

Congenital malformations of the biliary tract include duplications, atypical locations, absence or atresias and abnormal biliary connections. Duplication is present when two separate cavities and cystic ducts are formed. The paired cystic ducts may empty into the same common duct, or into different segments of the extrahepatic biliary system. This should not be confused with bilobed or bifid gallbladders with division of the cavity into two compartments by a longitudinal membraneous septum. In these a common peritoneal coat causes the radiologic picture of a single gallbladder.

The biliary sac may lie on the left side within the left lobe of the liver as an isolated defect with no evidence of situs inversus. Another rare anomaly is a subcutaneous or extraperitoneal gallbladder directly beneath the skin above the abdominal muscles, with no hernial sac. Such a case was reported by Bullard in an 81-year-old woman with a mass beneath her ribs diagnosed as a fascial sheath tumor. We saw a patient with the gallbladder situated in the uppermost aspect of the dome of the liver.

Congenital absence of the gallbladder is infrequent. It is a hereditary condition, often associated with other biliary tract anomalies. Nelson, Hatch and Jackson reported its incidence as 0.065 per cent of 18,350 necropsies. Oral cholecystography discloses only nonvisualization, and the diagnosis usually is made at operation. The condition becomes manifest in adult life with symptoms of cholecystitis and cholelithiasis. The association of gallstones is fairly common. Operative cholangiography is important in establishing the diagnosis.

Congenital atresia of the extrahepatic biliary tract sometimes is encountered in early infancy, usually with other abnormalities such as absence of all or portions of the bile ducts or atresia of the common duct. This is a developmental defect resulting from failure of the anlage of the liver and the bile duct to differentiate. The association of skeletal abnormalities with congenital biliary disorders was described by Levin, who observed osteomalacia in older children with increasing hepatic decompensation after long survival, and a picture of rickets in younger patients. The common feature is obstruction to the flow of bile, with consequent hepatic damage. Jaundice occurs from the first day of life to about the first month, and with incomplete obstructions a fluctuating jaundice is noted in an otherwise normal infant. Sometimes the condition is accompanied by complete absence of the interlobular bile ducts and marked proliferation of the periportal cholangioles, with pathologic changes not unlike those of infantile hepatitis or congenital intrahepatic biliary atresia. These infants may reach the age of 3 or 4 years.

The diagnosis of biliary atresia is established by operative cholangiography, an essential procedure in the investigation of infants with increasing obstructive jaundice.

A congenitally absent common bile duct was seen in a 54-year-old woman by Sachs. This patient had nonvisualization of the gallbladder 2 years before operation. At surgery the gallbladder was inflamed and dilated, and stones were present in the cystic duct. The cystic duct entered the duodenum, the common duct being absent. The hepatic ducts entered the gallbladder at its midportion. We recently encountered a patient with congenital absence of the cystic duct. The gallbladder opened directly into the common duct as a diverticulum.

CONGENITAL ABSENCE OF THE SPLEEN

A syndrome of multiple congenital anomalies of the heart and gastrointestinal tract with congenital absence of the spleen was reviewed recently by Aguilar, Stephens and Crane. Included among the cardiac malformations were septal defects, anomalous venous return, pulmonary stenosis or atresia and transposition of the great vessels. Accessory lobes of the lung, partial situs inversus of the abdominal viscera and abnormal mesenteric attachments were present as well. Twenty of the 27 patients died before 1 year. A hematologic disorder of persistent erythrocytic inclusions identified as Heinz bodies was noted. Bush and Ainger reported a case in which a correct antemortem diagnosis of congenital absence of the spleen with congenital heart disease was made on the basis of cyanotic heart disease with hematologic disorders similar to those found after splenectomy.

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Chapter 2

Neoplastic Diseases

INTRA-ABDOMINAL TUMORS

THE symptoms of intra-abdominal neoplastic disease are protean. These tumors may be asymptomatic for long intervals. Sooner or later, disorders appear which vary from insidious weakness and malaise to acute abdominal catastrophies. Often clinical manifestations of tumors of diverse origin are practically identical. Radiologic examination is one of the most reliable of our diagnostic methods.

NEOPLASMS OF THE STOMACH

Gastric tumors occur most often in the distal third. About 95 per cent are adenocarcinomas. Cancer of the middle and upper thirds of the stomach are less frequent. Although most patients are in their middle years, young people also are affected. The symptoms depend on the size of the growth, ulceration with involvement of the various coats, perforation and spread. Occasionally a rather hopeless attitude is assumed because of the supposition that these are incurable. This can be countered by sharpening our diagnostic acumen so that the disease is identified in its early stages, when something perhaps can be accomplished. The borderline between the normal and abnormal presents the radiologist his most challenging opportunity. The importance of a correct diagnosis almost appears to be inversely proportional to the size of the neoplasm. Certainly few large growths are overlooked, but small mucosal or submucosal tumors without ulceration or infiltration are far more difficult to identify.

Mesenchymal gastric tumors are infrequent. Included are leiomyomas and leiomyosarcomas of muscle origin, lymphomas of various lymphoid origin, neurinomas originating from nerve tissue, and various fibromas and lipomas. Those of lymphoid origin are more frequent. Lymphomas usually occur as part of a generalized disease, but may be primary in the stomach. Other disease processes, such as primary or secondary amyloidosis, or eosinophilic granulomas may simulate malignant disease. Metastatic deposits in the stomach are relatively uncommon.

Carcinomas of the stomach occur as spreading infiltrating lesions and as fungating papillary tumors which proliferate into the stomach and burrow through its walls to project into the abdominal cavity. Ulceration and pain as well as bleeding are relatively common, so that a clinical picture like that of ulcer frequently appears in younger people. As a rule, gastric hemorrhage is not profuse, but sometimes it may be exsanguinating. Kane, Meyer and Kozall found 673 cases of massive hemorrhage in a review of 28,400 necropsies. In 310 it was the cause of death, and in the others was regarded as contributory. Fatal hemorrhage occurred most frequently from esophageal varices. Gastric and duodenal ulcers were second and third respectively, and ruptured aneurysms into the gastrointestinal tract were fourth in incidence. Hemorrhage from carcinoma of the stomach was fifth in order.

NEOPLASMS OF THE CARDIA

Before proceeding with radiologic examination, each patient should be questioned as to history. Before permitting the patient to swallow the barium mixture, he should be asked if there had been any difficulty in swallowing. It is surprising how often this is overlooked in the absence of a major impairment. Fluoroscopic observation of the stomach gas bubble should precede the ingestion of barium. This sometimes provides the first indication of a fundal tumor even in the ab-

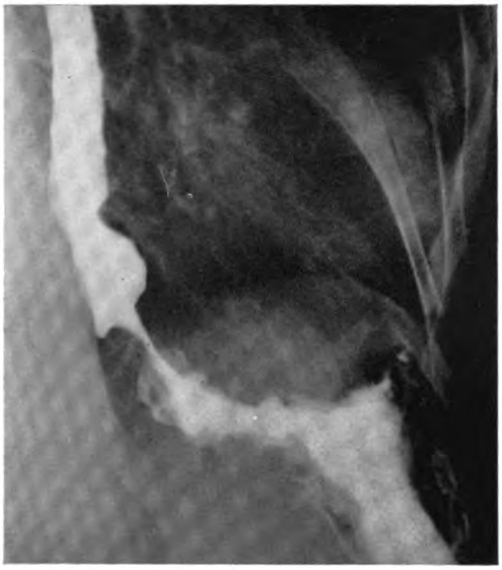


FIG. 53.—Carcinoma of the fundus separating the gastric cavity from the left diaphragm. Patient was a 77-year-old male.

sence of clinical complaints. The first swallow of barium may show a slight hesitation and displacement of the column as it enters the cardio-esophageal junction. Bulky tumors present as soft tissue masses projecting into the gas bubble. Often sufficient gas is present to permit such identification. If this is lacking, ingestion of a carbonated beverage readily provides enough stomach gas for contrast purposes. If the neoplasm extends above the cardio-esophageal junction the irregular thickening of the fundal convexity is apparent. Infiltrating tumors in this area without any real bulk are most difficult to identify. With perforation of the tumor through the gastric wall the space between the gas bubble and the left diaphragm is increased. There may be considerable bulk of tumor here, with invasion of the inferior surface of the diaphragm. Impairment of motion of the left diaphragm is not seen until really extensive invasion or marked bulk of tumor impedes its motion. This is a late manifestation. Extension into the retroperitoneal tissues causes forward displacement of the stomach as seen in oblique and lateral views. Invasion of the distal esophagus may cause marked delay in the passage of barium, sometimes associated with sufficient dilatation to mimic achalasia. In doubtful cases examination of the gas-filled stomach, or investigation after passage of a nasogastric tube and instillation of barium must be made to rule out a fundal neoplasm.

Tumors of the cardia are sometimes simulated by extraluminal masses. An aortic aneurysm bulging against the cardia at first glance may be confused with a tumor. Other changes such as calcification of the aneurysmal wall, or erosion of adjacent vertebræ serve to indicate the correct diagnosis. Enlargement of the left lobe of the liver as part of diffuse hepatomegaly or due to tumor may be indistinguishable from a gastric carcinoma radiologically. Occasionally gastric varices assume sufficient size to be tumorous. The identification of concomitant esophageal varices is helpful in making this diagnosis. Localized hypertrophy of rugal gastric folds also may assume tumorous proportions. Other possibilities include retroperitoneal tumors, pancreatic tumors and, rarely, tumors of the diaphragm. Occasionally an enlarged spleen impresses itself against the lateral and superior wall of the stomach simulating an intrinsic mass. Pronounced enlargement of the left ventricle bulging downwards has been mistaken for a fundal tumor, but fluoroscopic examination, preferably after the patient has taken a carbonated drink to distend the stomach, serves to clarify this condition.

Neoplasms of the middle third of the stomach are found generally along the lesser curvature, and extend to the posterior wall and anteriorly. Infrequently the greater curvature is the site of origin, as occurred in 7 of our random series of 120 cases. Usually there is no sharp line of delimitation between tumors of the cardia and pars media, and frequently both areas are involved simultaneously. The paucity of symptoms in large tumors often is surprising. Tumors of the pars media are frequently infiltrative, but bulky tumors also are found. Ulceration and bleeding are often present. The ulcers sometimes are quite small, but also reach considerable size. No radiologic characteristics serve to identify clearly benign from malignant ulcers. It is generally assumed that an ulcer of an intraluminal variety associated with extensive adjacent mural infiltration is

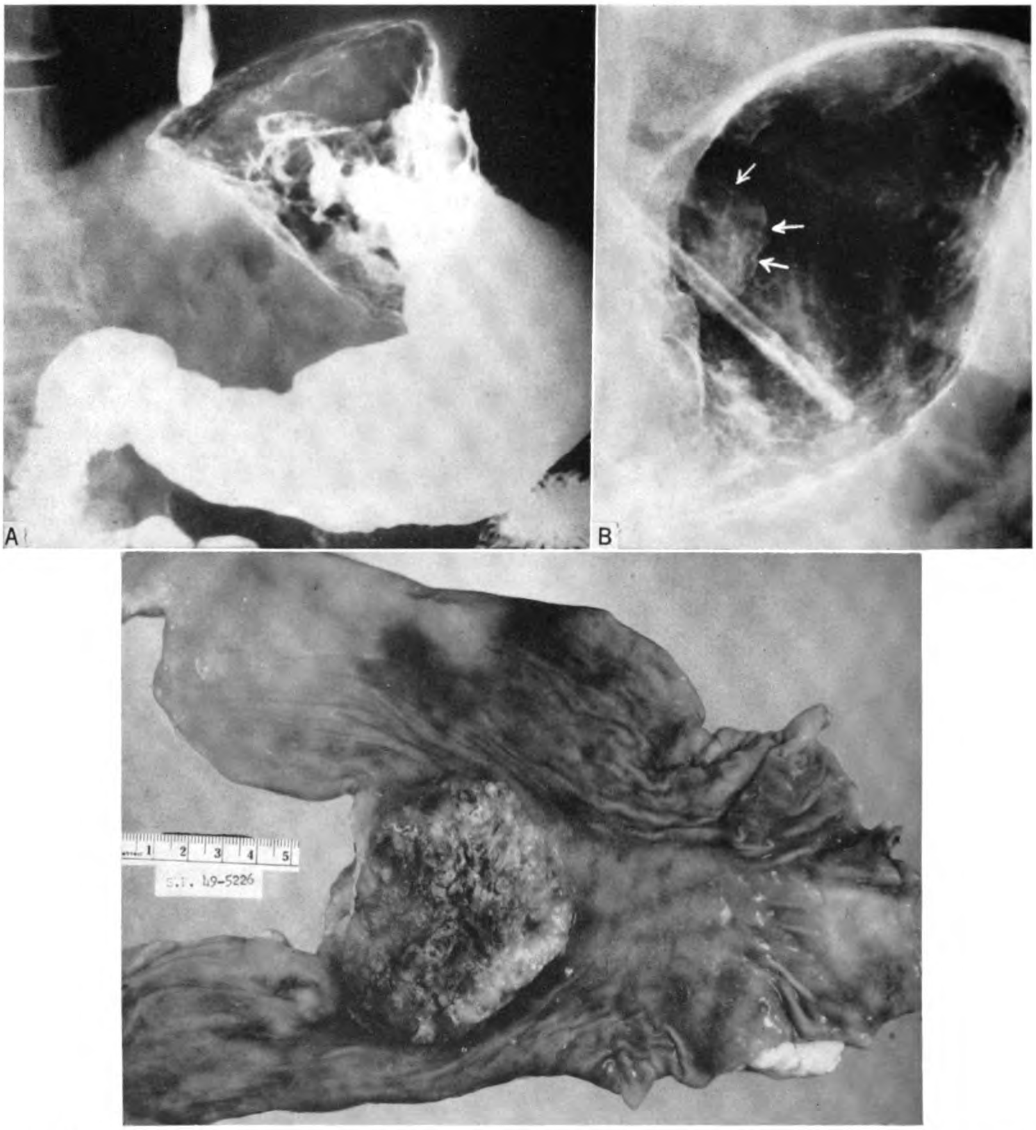


FIG. 54.—*A*, Carcinoma of the cardia in a 75-year-old man with no symptoms except hematemesis and melena. Right prone oblique fails to reveal the tumor.

B, Same patient after insufflation of gas into stomach through a nasogastric tube. A polypoid tumor is present above the esophageal opening.

C, Specimen of gastric carcinoma.

more likely to be malignant. However, a considerable diversity of opinion exists as to the value of signs such as convergence of folds towards a crater, undermining of ulcer borders, irregularity of the base and the rate of healing or the presence of a normal incisura in differentiating a benign from a malignant ulcer. Kirsh, in reviewing 142 histologically verified gastric ulcers, found 22 to be malignant and 120 benign. Radiating mucosal folds from the edges of the crater were found in 42 per cent of benign ulcers, but in none of the malignant ones. Abnormal folds and filling defects in the ulcer vicinity were ob-

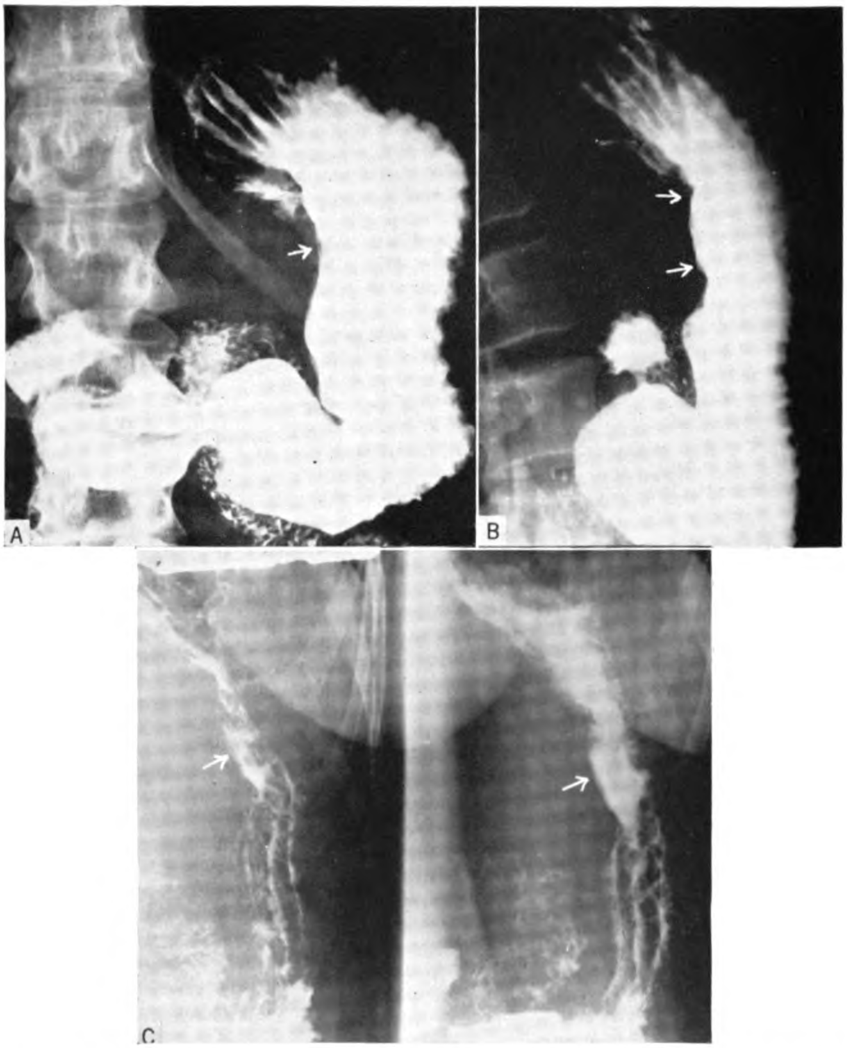


FIG. 55.—*A*, Carcinoma of the pars media, lesser curvature. Posteroanterior prone film reveals a questionable area of stiffening of the upper portion of the lesser curvature (arrows).

B, Same patient, supine right oblique view reveals the rigid area more clearly with shallow elongated ulcer crater (arrows).

C, Spot films made after first swallow of barium. The area of rigidity of the lesser curvature shows little mucosal change.

served in 85 per cent of malignant ulcers, but in not over 14 per cent of benign ulcers. Lloyd and Morris commented on the difficulty in differentiating malignant from benign ulcers. They found that intraluminal or extraluminal position of the crater with respect to the stomach wall, the size of the crater and the convergence of gastric folds were not entirely reliable. In some the folds stopped short of the ulcer margin, presumably due to a surrounding zone of infiltration, while in others the folds reached the ulcer edge without interruption.

The differentiation of benign from malignant ulcers has obvious therapeutic implications. Some hold that a patient with a benign ulcer is entitled to conservative therapy before operation is advised. However, Ravdin

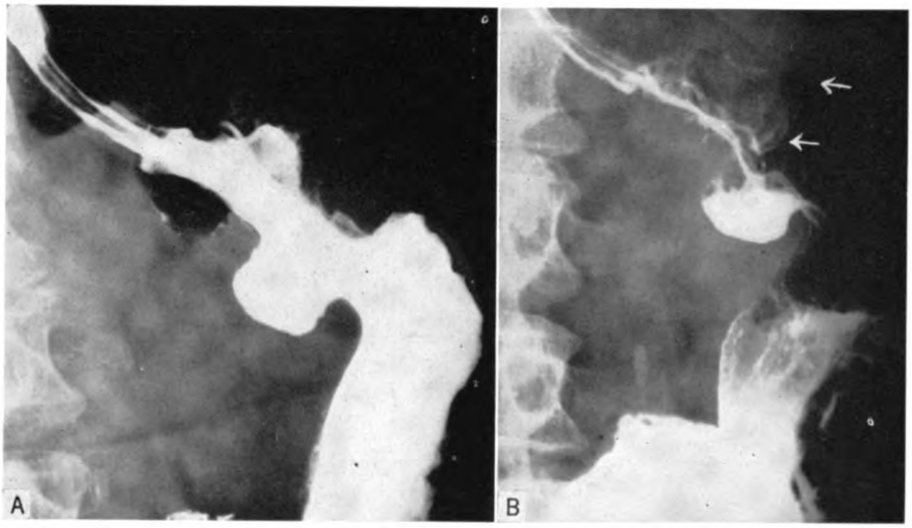
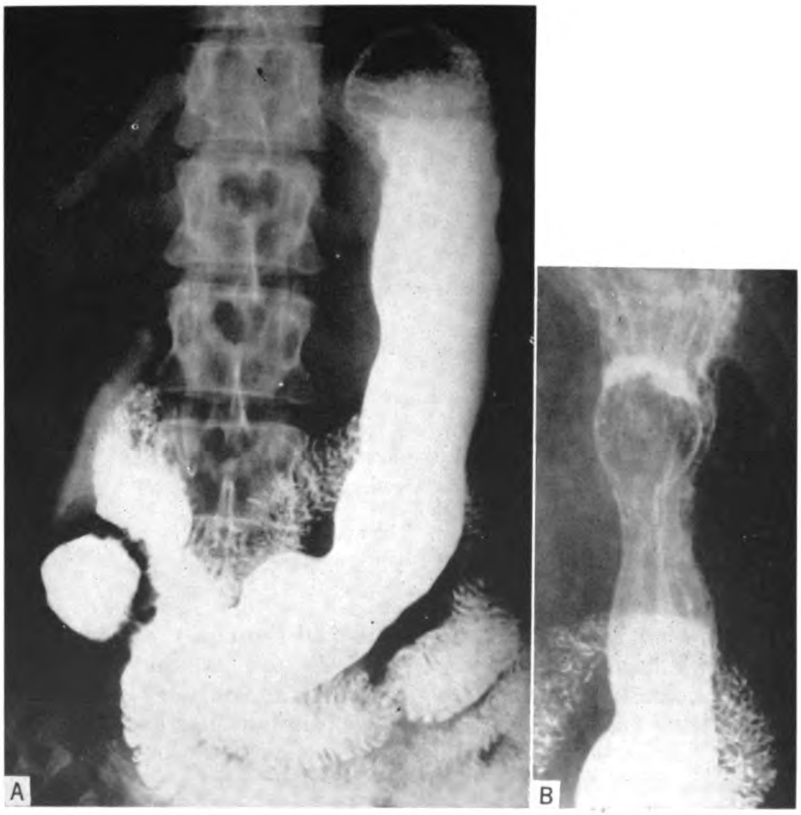


FIG. 56.—*A*, Penetrating carcinomatous ulcer of the lesser curvature in a 56-year-old woman. Right prone oblique view. The ulcer crater is beyond the gastric wall.

B, Same patient, erect posteroanterior position. The ulcer simulates a gastric diverticulum. The rigid area above the crater supports the diagnosis of a malignant ulcer (arrows).



FIG, 57.—*A*, Sessile carcinoma of pars media in a 60-year-old woman who complained only of weakness and weight loss. Posteroanterior prone films with filled stomach shows nothing pathological.

B, Same patient, spot film with compression reveals a circular filling defect on the posterior wall of the pars media. At operation a sessile polypoid adenocarcinoma was found.

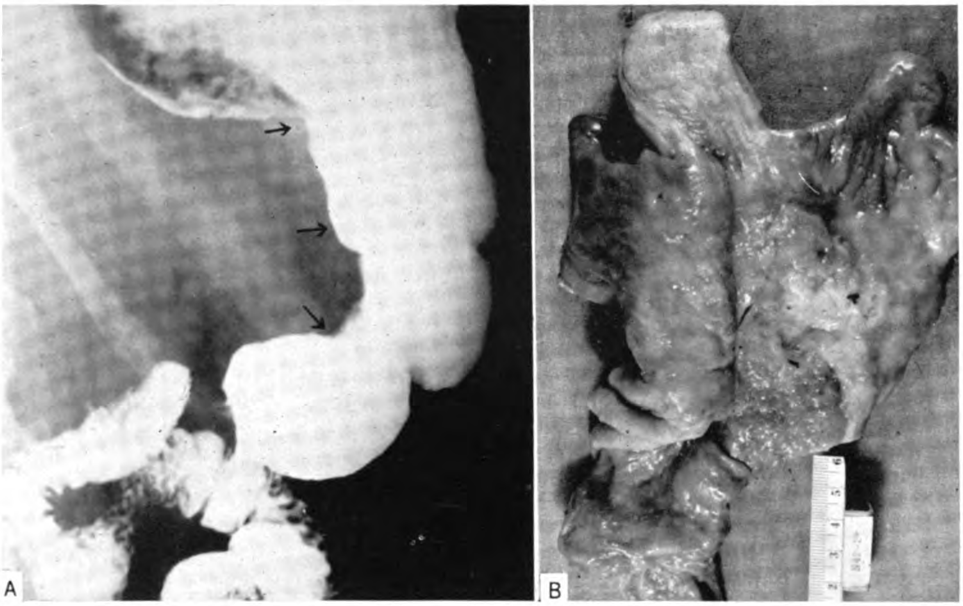


FIG. 58.—*A*, Scirrhous carcinoma of the pars media extending from lesser curvature to the posterior wall (arrows) in a 58-year-old woman.

B, Specimen of stomach with scirrhous carcinoma encircling the middle third.

and Horn asserted that since malignant ulcers cannot be accurately distinguished from benign ulcers preoperatively, all should be resected. In a review of 193 cases, only 43 per cent of 44 with a preoperative diagnosis of carcinomatous ulcer proved malignant. Of 99 cases with a preoperative diagnosis of benign ulcer, 11 had carcinomas. Another question is whether benign ulcers have malignant potential. Ravdin and Horn noted that 5 of their cases had definite evidence of carcinoma arising in primarily benign gastric ulcers, and estimate that this is possible in about 10 per cent of patients. Doll, in a review of perforative gastric carcinoma simulating ulcer, mentioned that even a biopsy was unreliable.

Occasionally perforation of a carcinomatous gastric ulcer is marked on plain abdominal roentgenograms by a pocket of gas in its vicinity. A bit of barium may collect at this site as well. Perforations are either sealed or not, the former being unassociated with generalized peritonitis, while in the latter peritonitis is evident as gastric contents enter the abdominal cavity.

Pyloric neoplasms are the most frequent of gastric tumors. The clinical picture is predominantly one of pain which often simulates peptic ulcer and sometimes gallbladder disease. Weakness, anorexia and vague gastrointestinal disturbances are common complaints, and later weight loss, vomiting and hematemesis appear. Any patient, particularly one in middle life, with persistent significant gastrointestinal complaints for more than a week or 10 days should be examined radiologically. Not infrequently a definite diagnosis cannot be made from a single examination, and there should be no hesitation in repeating the study.

Most antral neoplasms involve the lesser curvature. The tumor may vary from a tiny mucosal or submucosal rigidity to a lesion which envelopes the antrum and extends cephalad. Associated with this are mucosal aberrations which often are not specific. Indeed, the overlying mucosa and submucosa may be practically undisturbed. On the other hand, hypertrophy of the rugæ with diffuse inflammation and antral ulcers sometimes cannot be differentiated from carcinomas

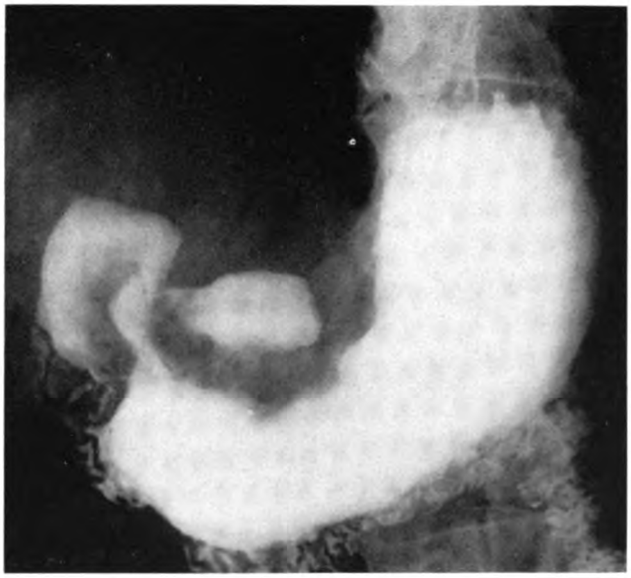


FIG. 59.—Perforating ulcerating adenocarcinoma of the lesser curvature in a 66-year-old man with abdominal pain, anorexia and weight loss. The tumor penetrated deep into the pancreas.

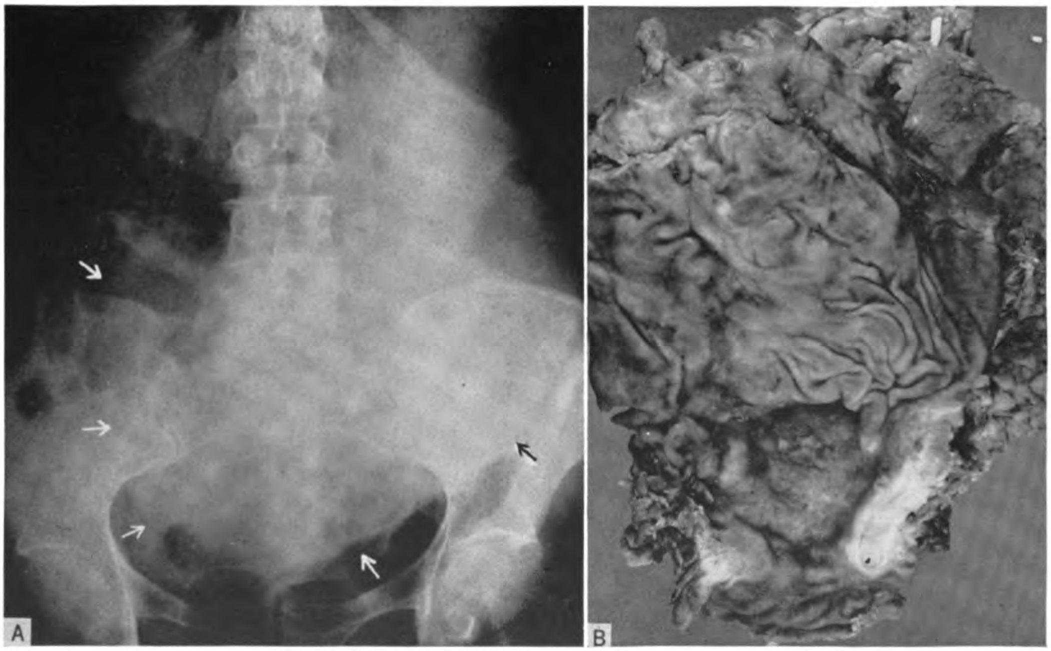


FIG. 60.—*A*, Marked dilatation of the stomach due to annular carcinoma of the pylorus in a 71-year-old man. The stomach practically fills the abdomen (arrows).
B, Specimen showing pyloric annular carcinoma.

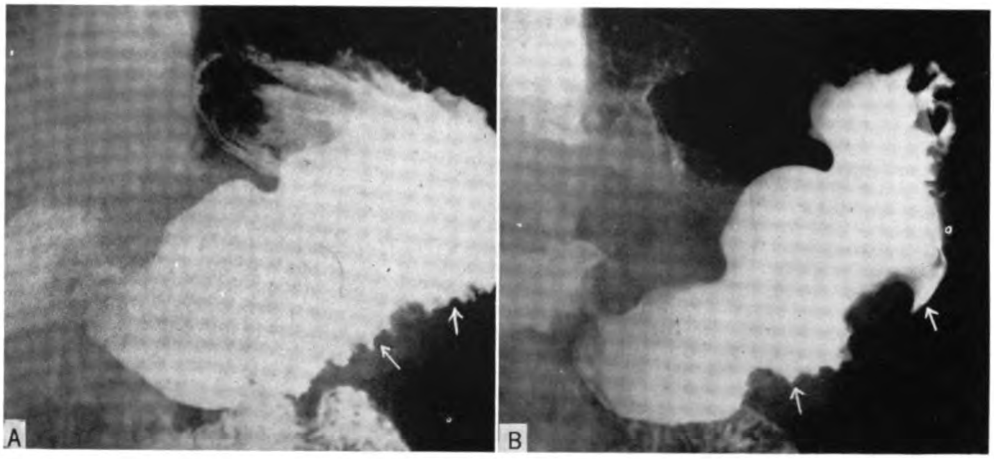


FIG. 61.—*A*, Scirrhous carcinoma of the greater curvature in a 74-year-old man (arrows). *B*, Same patient 6 weeks later. Note the progression of the tumor (arrows).

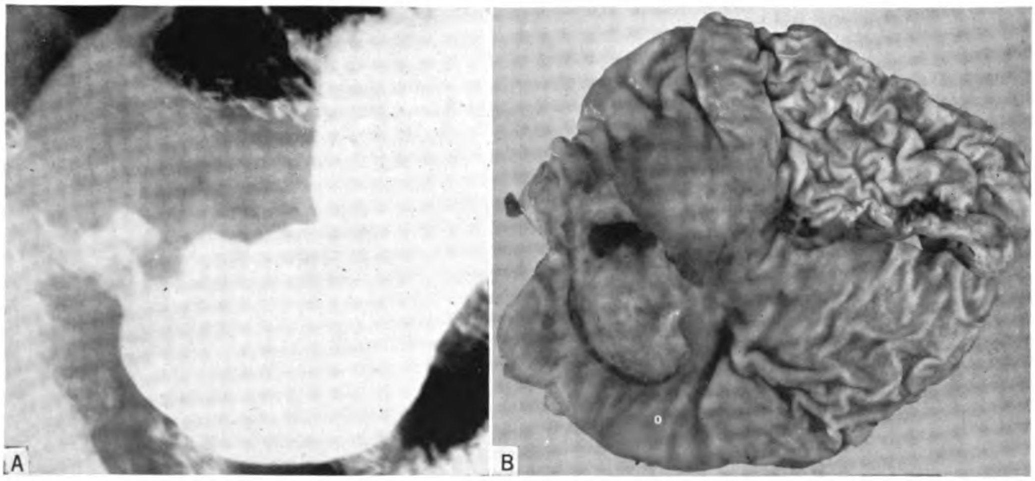


FIG. 62.—*A*, Carcinomatous ulcer projecting beyond gastric wall in a 62-year-old man with an 18-year history of epigastric distress relieved by food, and recent bleeding.
B, Same patient, specimen showing large carcinoma with penetrating ulcer.

either radiologically or by inspection of the gross specimen. The diagnosis of an obvious gastric cancer requires no great skill, but the identification of a small, flat early malignancy is a credit to the observer.

Gastric malignancies encircling the pylorus produce a "napkin-ring" lesion, often with little mucosal change. A deceptive picture is associated with this, particularly when the lesion is not obstructive. Eventually, however obstruction is followed by gastric retention and dilatation which sometimes cannot be differentiated from that of calloused ulcer or infrequently, benign hypertrophy of the pyloric muscle.

Intraluminal polypoid tumors present a characteristic fungating irregularity. The deformity with these tumors portrays the available space left in the stomach, the mass itself often being much larger than anticipated from inspection of the filled stomach.

The radiologic differentiation of benign antral ulcers from malignancy is difficult. Occasionally the lesion is considered benign

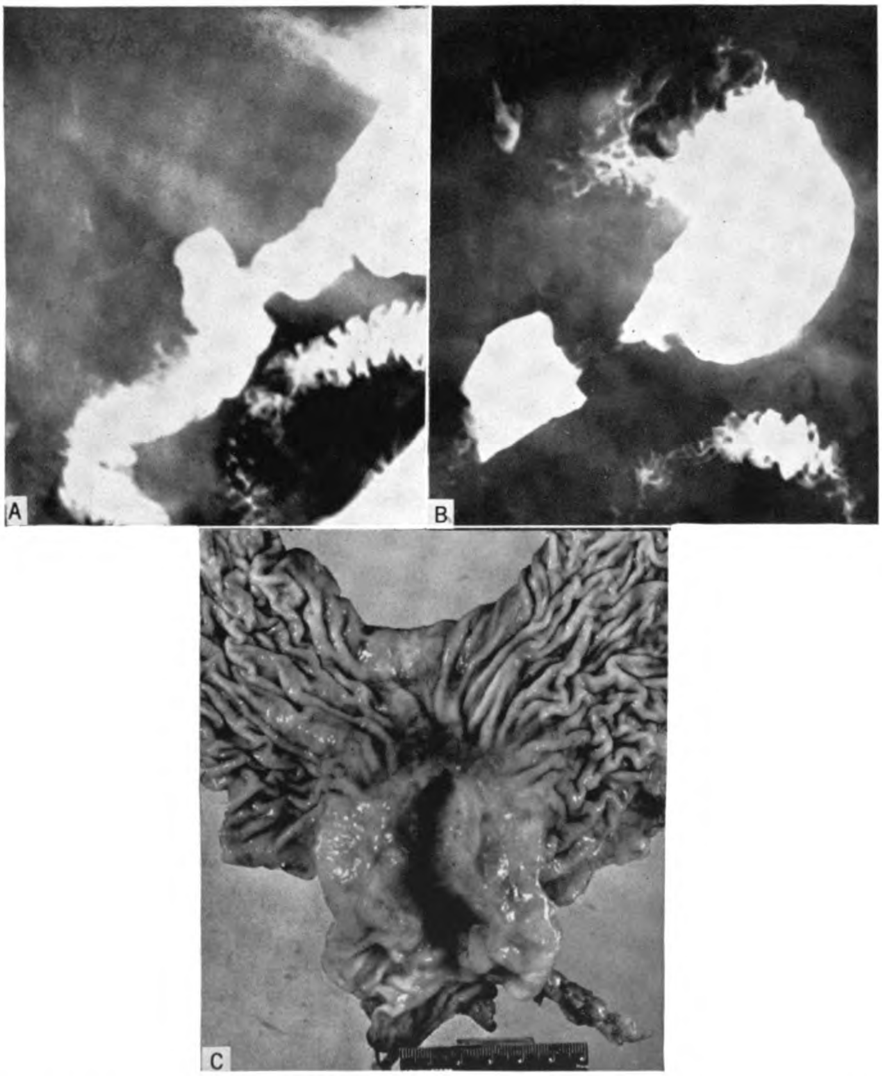


FIG. 63.—*A*, Pyloric carcinoma in a 56-year-old man. The antrum is narrowed and appears to be tubular.

B, Same patient, same position about 5 minutes later. Spasm of the pylorus has altered its configuration to a tapered structure separated from the antral ring by a radiolucent area.

C, Same patient, specimen showing that the prepyloric area is relatively less infiltrated with tumor than the remainder of the antrum.

if the irregularity vanishes with medical treatment. However, in our experience recently there were 6 cases in which a gastric ulcer supposedly was healed, only to recur as a cancer 6 months to a year later. As with ulcers elsewhere in the stomach, great caution should be exercised in making what actually is a histologic diagnosis on the basis of radiographic changes.

Occasionally pyloric malignancies originate on the greater curvature as infiltrating or fungating tumors, together with ulcers. While benign curvature ulcers are reported quite often, it is better to settle the question by histologic examination of the specimen than to rely on radiologic criteria.

Gastric cancers extend to adjacent tissues by direct extension, and often metastasize

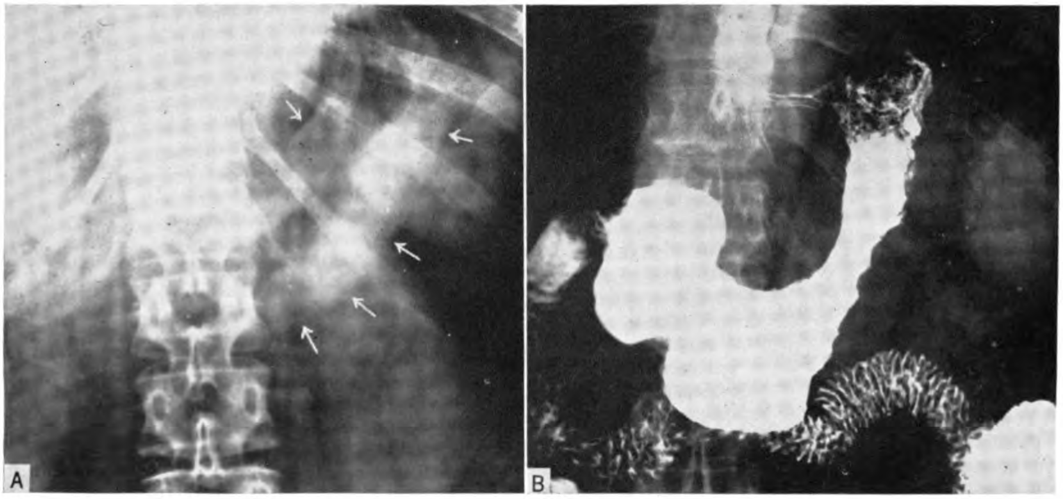


FIG. 64.—*A*, Linitis plastica in a 53-year-old woman with anorexia and weight loss. Scout abdominal films reveals thickened, tumefied gastric walls (arrows).

B, Same patient, anteroposterior roentgenogram of stomach revealing gastric contour conforming with that seen on scout film.

to the liver, lymph nodes and peritoneal cavity. Obstructive jaundice may follow invasion of lymph nodes in the porta hepatis or the liver. Infrequently, gastric tumors extend to the adjacent transverse colon or to the spleen and retroperitoneal regions. In 2 recent cases the initial complaints were referable to an obstruction in the midtransverse colon. The gastric origin of this was not known until operation after a barium enema had disclosed what appeared to be a transverse colon neoplasm.

Golden and Stout noted gastric carcinomas arising in the mucosa, which remained and grew in the mucosa and submucosa for some time before becoming invasive. They considered these theoretically curable if identified in time, and regarded them as appearing not infrequently in the lower half of the stomach. In a review of 31 cases, open ulcers were associated with this variety of superficial gastric carcinoma in 26. The cancer had developed around an open ulcer in 12 cases. These authors also encountered difficulty in differentiating antral ulcers from spasms with gastritis.

Occasionally intense pyloric spasm is seen which simulates malignant disease. The radiographic changes are persistent, but no tumor is found at operation. Jenkinson and Hamernick described 7 cases with a radiologic diagnosis of cancer, but nothing abnormal was found at operation. On analysis of the clinical picture, they observed that none of these patients had symptoms of carcinoma commensurate with the radiologic changes.

Another infrequent tumor is the villous tumor of the stomach. Walk reported that only 51 such cases had been described, and added 2 of his own. These are cauliflower-like papillomas which should be differentiated from benign polyps. Although both are adenomas, the villous tumor has a greater malignant potential. Radiographically villous tumors present a rather characteristic appearance described as "soap-bubble." Epigastric pain, blood loss and diarrhea often are present, and usually a severe anemia dominates the picture. Apparently the malignant capacity of these tumors varies directly with their size, the smaller ones tending to be benign. Most malignant changes occur in the stalk.

Tumors involving the entire stomach are not uncommon. As a rule these are scirrhous and relatively indolent in their development. Radiologically the stomach is represented by

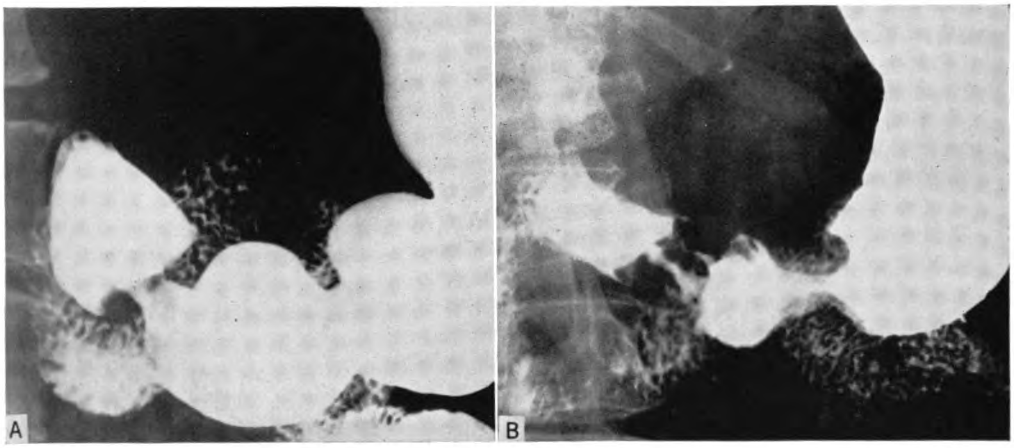


FIG. 65.—*A*, Annular pyloric carcinoma in a 63-year-old man with tarry stools. No pain, weight loss or vomiting.

B, Same patient, a few moments later, showing variability in the roentgenologic appearance. At operation an annular carcinoma with a central ulcer was found.

a cavity encased in a rigid, thick wall. In 3 of our cases the stomach walls were so thickened that they could be identified on plain abdominal roentgenograms in contrast to the intragastric air. When filled with barium, the leather-bottle appearance with a markedly altered mucosal pattern is diagnostic. In some the mucosa is practically obliterated, while in others the pattern is stippled or granular with nodular projections.

Occasionally a localized adenocarcinoma of the stomach simulates a benign adenomatous polyp, a heterotopic pancreatic implant or an extramucosal mass. A reticular mucosal pattern suggests the presence of infiltration or nodulation, which supports the diagnosis of a malignant tumor. As a rule benign tumors tend to be sharply circumscribed with normal mucosal patterns. A localized hypertrophic gastritis also may simulate a nodular carcinoma.

Rarely carcinomas of the stomach contain radiologically demonstrable calcifications. Kendig, Gaspar, Secrest and Shackford noted that a total of 9 such cases had been reported, of which 7 were verified, and they reported another in a 27-year-old female. Mucinous degeneration within the tumor was present in several cases.

Infrequently multiple gastric tumors are found. These may be dissimilar, as observed in 3 cases of our cases. In 1 a benign polyp well separated from a fundal carcinoma was present. In another, a fundal tumor was accompanied by an antral lymphoblastoma. In the third a pars media carcinoma was associated with heterotopic pancreatic tissue in the antrum and duodenum.

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GASTRIC POLYPS

Any pedunculated newgrowth or hyperplasia arising from the gastric mucosa is regarded as a polyp. Many are of epithelial origin, and occur as single or multiple lesions anywhere in the stomach as pedunculated or sessile growths varying in diameter from a few millimeters to several centimeters. These may be simple mucosal polyps or contain glandular and muscular elements. Polyps also arise from a common base, resembling closely packed orderly rows of polypoid tissue which simulate, or perhaps may be, villous polyps. Infrequently, the entire stomach is diffusely covered by polyps from cardia to pylorus.

A diversity of opinion exists as to whether polyps are premalignant lesions. Colp and Weinstein reported 2 cases in which carcinomas developed. Various reports indicate a possible malignant alteration in from 6 to 50 per cent of cases, although the latter figure is disputed.

Polyps of the stomach are relatively infrequent. Overgaard found 17 in the stomach and duodenum in approximately 3600 gastric examinations, of which 10 were single and 7 multiple. Nine had gastric polyps, 5 were duodenal, and in 3 cases they were found both in the stomach and duodenum. In only 1 case was surgical confirmation obtained. Ten showed no change on radiologic examinations from 7 to 11 years after the initial study. Overgaard believed that gastric polyps were not necessarily precancerous, and suggested that treatment be conservative unless bleeding or obstruction were present. In a review of 8735 necropsies, Yarnis, Marshak and Friedman encountered 73 gastric polyps diagnosed *in vivo*, and 30 more were found at autopsy. Malignant transformation was found in only 2 of their cases.

Gastric polyps often go unrecognized unless pain or bleeding brings the patient to the physician. Indeed, the identification of a gastric polyp can mask other more serious disease. This diagnosis should not be accepted when complaints are at variance with the radiologic observations.

Gastric polyps occur on stalks or as broad

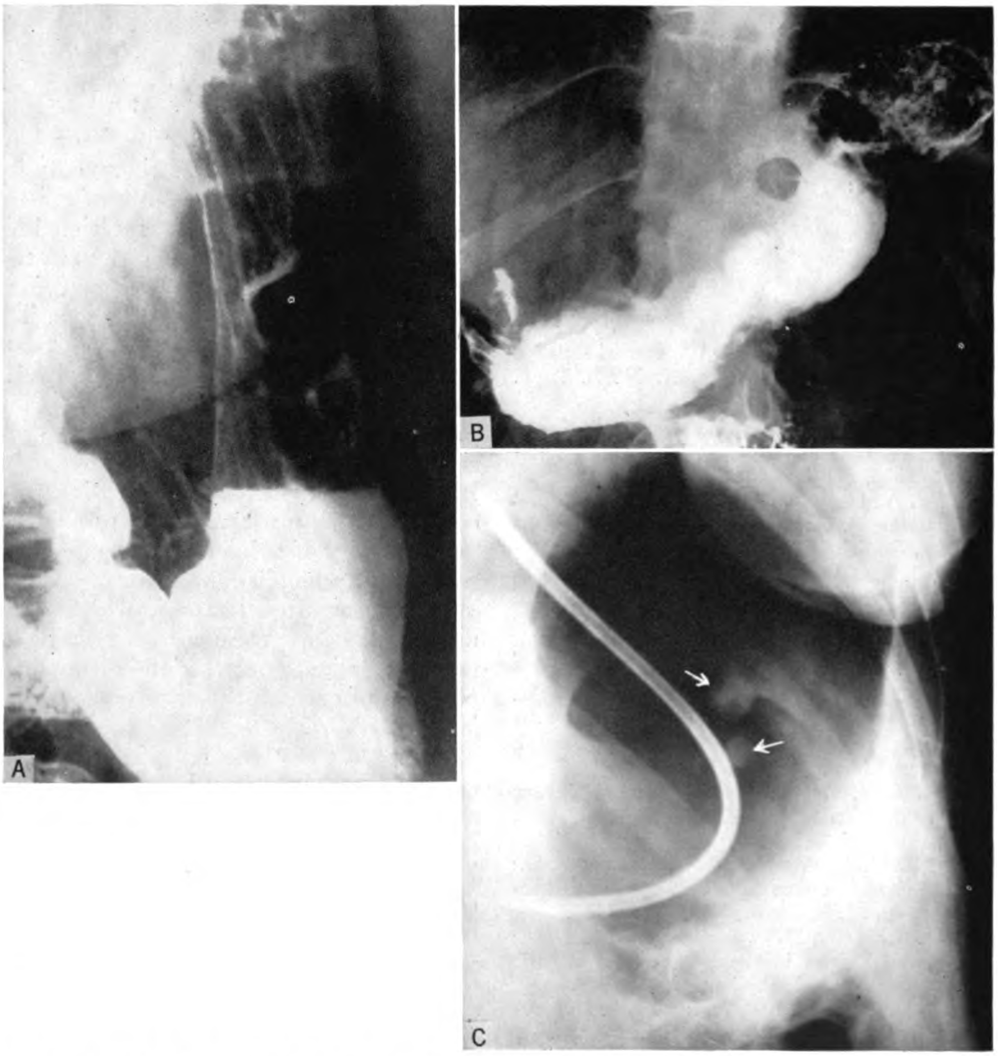

FIG. 66.—*A*, Multiple gastric polyps in a 57-year-old woman with chronic gastric complaints for many years. No bleeding episodes. Film made in August, 1946, reveals 2 polyps at least (arrows).
B, Same patient, 5 years later. No change in size of polyps or the patient's symptoms.
C, Same patient, air contrast study.

based sessile tumors. In our 15 cases, the polyps varied from 1 cm. in diameter to projecting masses more than 7 cm. wide. Four had multiple polyps. All of these cases were verified at operation. In 6 the presenting symptom was gastric hemorrhage due to ulceration of the dome of the polyp. One had polyps which practically filled the stomach with masses of widely varying size. Some were entirely bengin, while others had definite malignant degeneration. In 3 additional cases malignant degenerative changes were seen.

The radiologic identification of these lesions requires careful investigation of the stomach wall and mucosa with compression and air contrast examinations. In differential diagnosis particles of food or air bubbles sometimes cause difficulty. Hypertrophied rugal folds may appear much like small adenomas. An interesting complication occasionally observed is gastroduodenal intus-

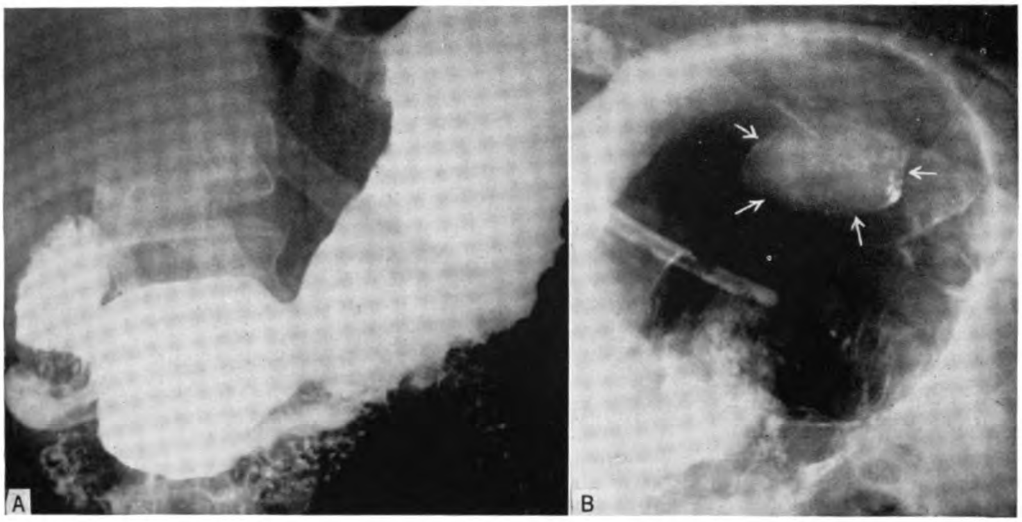

FIG. 67.—*A*, Bleeding adenomatous polyp in cardia of a 63-year-old man with recurrent gastric hemorrhage. Posteroanterior prone film fails to reveal the polyp, which is covered by barium.

B, Same patient, air contrast study shows large polyp hanging from cardia. At operation the tumor measured 5 × 4 × 2 centimeters.

susception with the polyp acting as the head. Intraduodenal prolapse of antral mucosa should not be mistaken for polypoid changes. Other lesions which simulate polyps are heterotopic implants of pancreas. In our own material there were 3 cases of fibrosarcoma of the greater curvature in which the overlying mucosa was intact. The diagnosis before operation was gastric polyp in all 3 cases.

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MESENCHYMAL GASTRIC TUMORS

Leiomyomas probably are the most frequent of benign gastric neoplasms. Originating in the smooth muscle, these project into the lumen of the bowel simulating a smooth-walled mass. They may infiltrate the wall, or project well beyond the confines of the stomach in a predominantly extragastric form. Not infrequently leiomyomas are both intra- and extra-gastric. These tumors vary from a few millimeters to masses which practically fill the abdomen. Most are of relatively moderate size, and many go undiagnosed until identified incidentally at necropsy. The age incidence is from about 13 years to old age, mainly in the third to fifth decades. They occur anywheres in the stomach, but are more frequent in the lower half. Usually these patients have few symptoms until ulceration with pain and bleeding appear. The bleeding usually is slow, but can be profuse. Occasionally vague and indistinct complaints such as nausea, weakness and vomiting are present. If the tumor is properly situated it can produce pyloric obstruction, and when sufficiently large, can be palpated. A rather infrequent complication is gastroduodenal intussusception with acute gastric obstruction. If the lesion is small, intussusception may be recurrent. The diagnosis then can be made only during an acute episode.

Leiomyomas undergoing malignant degen-

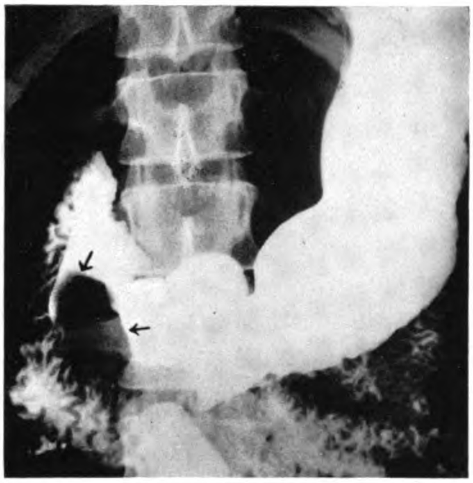

FIG. 68.—Fibrosarcoma in the antrum of a 28-year-old woman with gastric bleeding. Mucosa found to be intact over the tumor except for one ulcerated area which was bleeding. Tumor extended into the muscularis but not into the serosa.

eration become leiomyosarcomas, which cannot be distinguished from leiomyomas radiologically. These are relatively frequent, constituting about 10 per cent of gastric sarcomas. While they tend to remain encapsulated, they often recur and metastasize to regional lymph nodes, the liver, omentum and the peritoneal cavity.

The radiologic diagnosis of leiomyomas and leiomyosarcomas is sometimes established on scout films if the extragastric mass is sufficiently large. This may simulate splenomegaly if situated in the left upper abdomen. Mottled scattered calcifications within a leiomyomatous tumor may lead to a correct diagnosis. When sufficiently large the adjacent viscera is displaced considerably. The configuration of the gastric walls reflect the intra-gastric and extragastric masses. Sometimes the diagnosis is more readily made if a carbonated beverage is given and films made immediately thereafter.

Small leiomyomas are sometimes identified by relatively small, discrete circumscribed filling defects with flattening of the overlying mucosa. The surrounding folds, however, remain normal. If the mass is not too large normal peristalsis persists. A rather abrupt angle is often formed at the junction of the tumor base and the uninvolved gastric wall. If ulceration takes place, barium accumulates and is retained in the crater at times. Sometimes ulcers are so deep that they assume the proportions of diverticula.

While leiomyomas often simulate sessile polyps, they sometimes infiltrate and project into the stomach like carcinomas. In 1 of our cases a diagnosis of carcinoma of the pancreas had been made, but at necropsy a leiomyosarcoma was found to have penetraded the stomach and massively infiltrated the pancreas. Retroperitoneal and intra-abdominal masses also resemble these tumors. In reviewing our 8 cases, there were 2 in which a diagnosis of leiomyoma or leiomyosarcoma had been suggested radiologically. In the others the lesion could not be differentiated from carcinoma of the stomach or pancreatic tumor.

Occasionally a small mural tumor of the stomach is due to adenomyosis. Such a case was reported by Campbell, who observed a small filling defect in the pylorus with intact mucosa. On pathologic examination a small nipple-like structure was observed projecting from the mucosa. The orifice of a small duct was present in the center of the papilla, admitting a fine probe. The condition was diagnosed as adenomyosis of congenital heterotopic origin. The wall of this diverticulum was lined with gastric mucosa, and some pancreatic tissue also was present. Patients with this lesion may have symptoms like those of peptic ulcer, and relief can be obtained by removing the mass.

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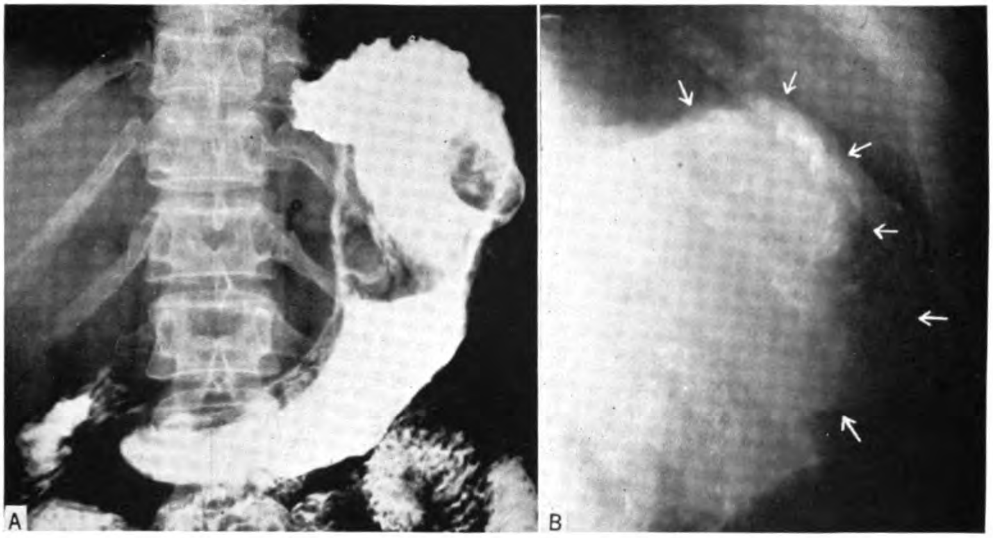

FIG. 69.—*A*, Leiomyosarcoma of stomach in a 49-year-old woman with weight loss, anorexia and vomiting. *B*, Same patient, air contrast study in lateral view shows a large mass impinging against the posterior wall, extending dorsally. At necropsy a large mass passing from the posterior wall of the stomach into the pancreas was found. The mass had ulcerated through the gastric mucosa.

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LYMPHOID TUMORS

No attempt will be made to differentiate such tumors radiologically. Histologically, these include Hodgkin's disease, lymphosarcoma, reticulum cell sarcoma and leukemia. The cellular structure of these growths do not alter their general radiologic appearance. Lymphomas are relatively uncommon, but they do constitute a large proportion of rare gastric neoplasms. Usually they occur as part of a diffuse process, but occasionally are limited to the stomach. Sometimes the picture is overshadowed by pain like that of peptic ulcer. Anemia, weight loss and achlohydria all occur. Pyloric tumors may achieve sufficient size to cause partial obstruction. Clinically, and usually radiologically, they cannot be told apart accurately from carcinomas.

Lymphomas occur mostly in young and middle aged people. As a rule, ulcerations in lymphomas are rather superficial and cause little necrosis. Perforation is relatively uncommon. According to Spellberg and Zivin, bleeding is a fairly common symptoms, producing hematemesis or hematemesis with melena.

Gastric lymphomas usually arise from the submucosal lymphoid tissue, and infiltrate the mucosa and muscularis. The pylorus is the most common site of origin, but gastric lymphomas are found anywhere in the stomach. As the lymphoid hyperplasia proceeds, there is gradual replacement of normal lymphoid tissue, with infiltration of the walls and extension to the serosa. Later Pel-Ebstein fever and cachexia appear. As a rule

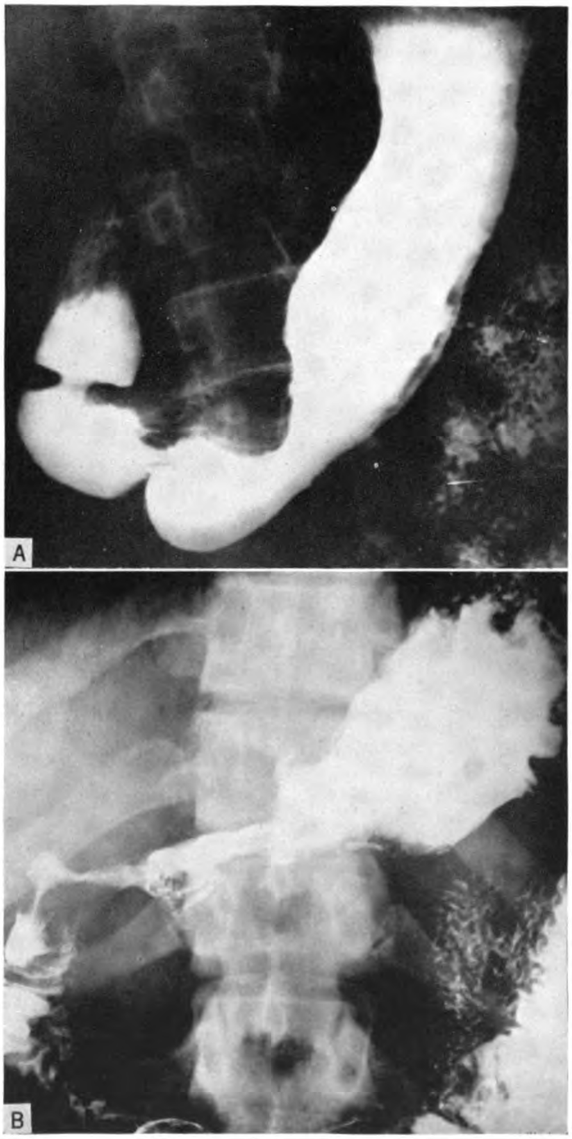

FIG. 70.—*A*, Localized submucosal reticulum cell sarcoma of the lesser curvature in a 47-year-old man. No gastric symptoms were present. The examination was performed as part of an investigation for midback pain.

B, Lymphosarcoma of pyloric antrum of infiltrative type in a 30-year-old man with an epigastric mass, midabdominal pain relieved by food, and weight loss of 20 pounds.

the blood picture is unchanged except in cases of leukemia. Extensive lymphomatous infiltration produces a leather-bottle stomach. With infiltrating tumors, peristaltic activity may persist. Mucosal nodular and polypoid changes often are prominent. Sometimes the tumor extends extrinsically to involve the adjacent viscera, particularly the lymph nodes. In the early stages of the disease, the radiologic changes may be minimal. Later gastric ulcers appear, some of which are large. The accuracy of preoperative diagnosis of gastric lymphomas is small.

Occasionally a pattern of markedly en-

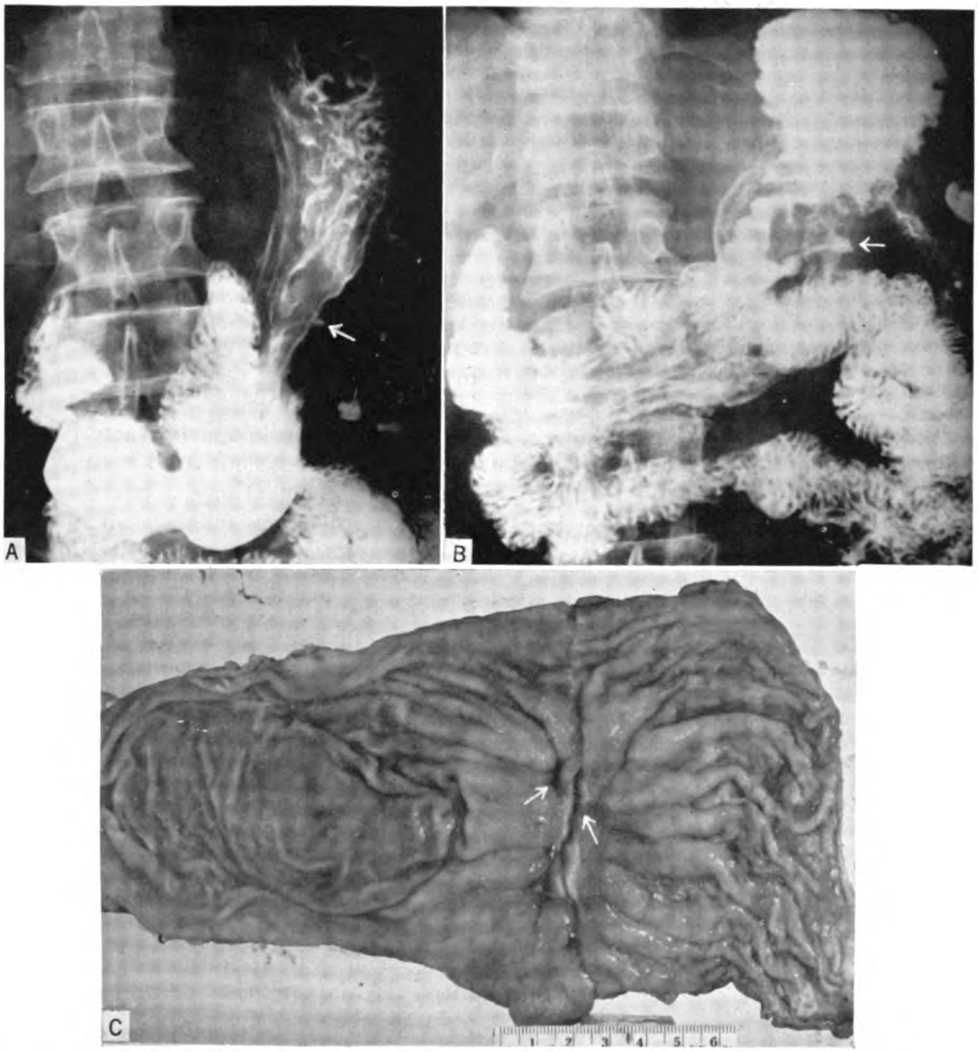

FIG. 71.—*A*, Posterior wall ulcer extending to greater curvature in a 69-year-old man with lymphosarcoma. Occasional abdominal pain and several episodes of melena. The crater (arrows) is in the line of the greater curvature wall.

B, Same patient, supine anteroposterior view. The ulcer has a double crater, one on the greater curvature and the other on the posterior wall (arrows).

C, Operative specimen showing double ulcer and hypertrophied rugæ.

larged rugal folds is observed. This is attributed to diffuse submucosal infiltration with lymphoma, and appears to be somewhat more common with leukemia. No destruction of the gastric wall is produced by this condition. Marked difficulty in differentiating this from benign rugal hypertrophy is not uncommon. Nodular lymphomas may simulate gastric polyposis.

In our own material there were 15 cases of gastric lymphoma. Histologically, 3 were lymphosarcomas, 3 reticulum cell sarcomas, 3 had leukemic infiltrations, and the others had Hodgkin's disease. This group is of interest in that 12 had lesions localized only to the stomach. Their ages varied from 30 to 70 years, and in most of the patients the initial symptoms were pain and gastric bleeding. Vomiting and regurgitation also were present. A large mass was palpable in 7 cases. At operation extension of tumor through the gastric wall was observed in 5, with infiltration of the mesentery and omentum. The radiologic changes varied from a localized nodular mass in the cardia to complete replacement of the stomach wall which the surgeon and pathologist first diagnosed as scirrhous carcinoma.

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Gastric lipomas are included with mesenchymal tumors. These are relatively infrequent, usually benign newgrowths. However, they do ulcerate and bleed. Usually they are well demarcated, producing sharply-circumscribed filling defects. When on a pedicle in the pylorus they may cause gastroduodenal invagination. At times these tumors are large enough to cause gastric obstruction. An important radiologic characteristic is radiolucency of the mass due to its fatty component. Sometimes a gastric dermoid cyst presents a similar appearance.

Tumors originating from the intramural gastric nerve structures also produce localized filling defects which cannot be differentiated from polyps. These neurogenic tumors have a malignant potential, and may bleed because of central ulceration of the overlying mucosa.

Another rare neoplasm of the stomach is the *plasma cell tumor*. These may occur without osseous involvement. The diagnosis is established only at operation.

Hemangioma of the stomach is occasionally identified by phleboliths within the mass. These tumors are of the capillary or cavernous variety, and occur singly or in multiple form. Hematemesis, bleeding and epigastric pain are the chief symptoms.

Rarely *Kaposi's disease* presents itself as a gastric lesion. Such a case with pain and hematemesis was reported by Grove, who stated that visceral involvement occurs in about 10 per cent of cases, particularly late in the disease. The gastrointestinal tract is involved most often, but lesions have been found in every organ. He reported a case in which the gastric lesions overshadowed those of the skin. This patient had multiple masses in the mucosal and submucosal.

Another infrequent gastric tumor of epithelial origin is the *carcinoid*. While these occur more often in the appendix and ileum, they also are found in the stomach as relatively smooth masses which simulate other nodular lesions. Ulceration and bleeding occur, and the initial symptom may be melena. These tumors arise from the argentaffin cells deep in the mucosal glands.

Metastatic tumors of the stomach also produce polypoid deformities. Most are from the breast or from melanomas. The correct diagnosis was made in 1 of our patients who had numerous circular deposits in the submucosa and in the duodenum.

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DUODENAL TUMORS

The greatest number of duodenal tumors are adenocarcinomas. The remainder are of mesenchymal origin, such as leiomyomas and leiomyosarcomas, of lymphomatous origin and carcinoids. Benign tumors such as polyps are infrequent. In a review of 25,032 necropsies Burgerman, Baggenstoss and Cain found 31 primary malignant tumors of the duodenum. Twenty-seven were adenocarcinomas, 2 leiomyosarcomas, 1 reticulum cell sarcoma and 1 lymphosarcoma. The occurrence of benign duodenal tumors was reported by Hoffman and Grayzel as 14 out of 64,300 surgical specimens and 4,480 necropsies. Barclay and Kent reported 0.19 per cent of all gastrointestinal carcinomas as primary in the duodenum. However, duodenal carcinomas constituted about one-third of all small intestinal cancers.

Duodenal carcinomas occur most frequently in the bulb or the suprapapillary portion of the loop. About one-third occur below the papilla of Vater, extending in some instances to the proximal jejunum. Duodenal cancers are found as predominantly annular constricting lesions, as diffusely infiltrating tumors extending for considerable distances, or as scirrhous lesions. Some are fungating and extend into the lumen, others are ulcerating and produce jagged irregular deformities. Extension into the adjacent soft tissues fixing the duodenum is frequent. With progression, necrosis and fistula formation to the colon, the retroperitoneal and paraduodenal areas takes place. Colwell found 22 such cases in the literature, and reported 1 in which a barium enema flowed from the hepatic flexure into the second portion of the duodenum and then into the stomach.

The diagnosis of duodenal carcinoma can only be suggested clinically, and requires radiologic confirmation. It is a disease of middle life, occurring more frequently in males. Duodenal ulcer or gallbladder disease are simulated, with pain, distress, weakness, weight loss and jaundice. Bleeding, sometimes profuse, occurs when the tumor ulcerates. Obstructive duodenal carcinoma produces gastric dilatation. Anemia is frequent. Sometimes no symptoms appear until the tumor has metastasized to regional lymph nodes, the liver or retroperitoneally.

The radiographic appearance of duodenal carcinoma reflects the gross pathologic changes. Where the tumor is infiltrative, the

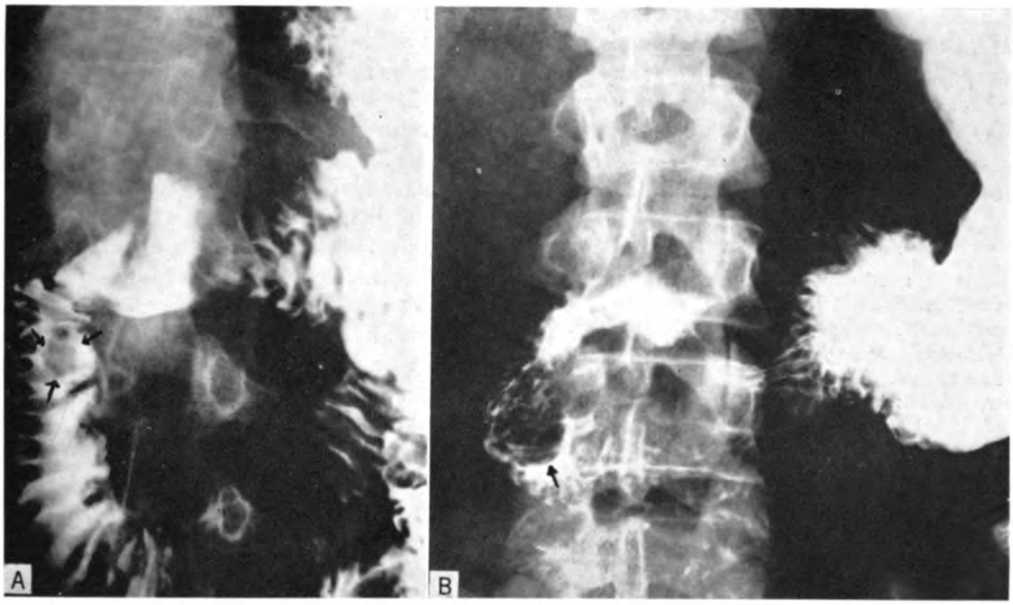

FIG. 72.—*A*, Submucosal mass in suprapapillary area of duodenum (arrows) in a 39-year-old man with cirrhosis of the liver and gastric bleeding. Postmortem examination revealed this to be a blind remnant of a ductal orifice, presumably a pancreatic duct. No symptoms were attributed to this.

B, Benign adenoma of descending duodenum (arrow) in a 45-year-old woman causing exsanguinating hemorrhage.

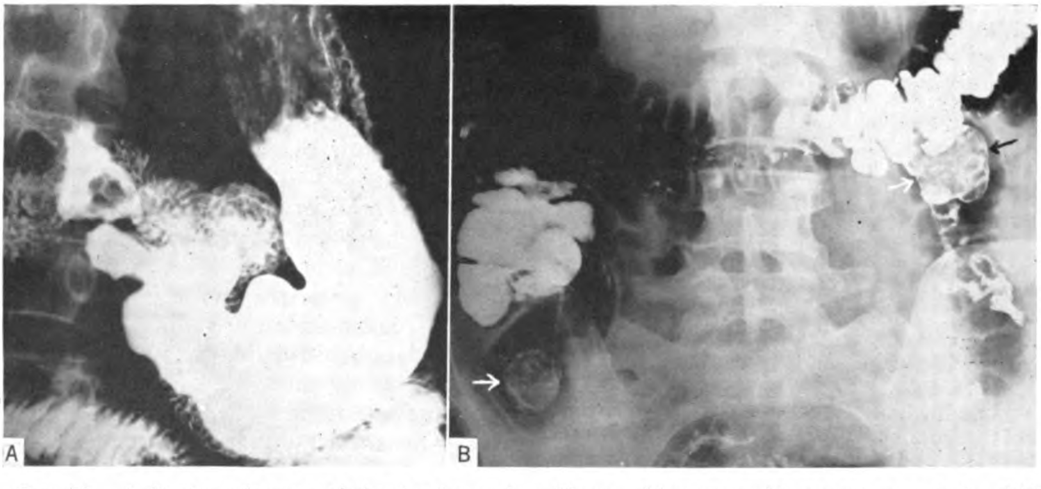

FIG. 73.—*A*, Benign adenoma of the duodenum in a 35-year-old woman with secondary anemia which responded to medical treatment.

B, Same patient, showing similar polyps in the terminal ileum (arrows) and in the descending colon (arrows).

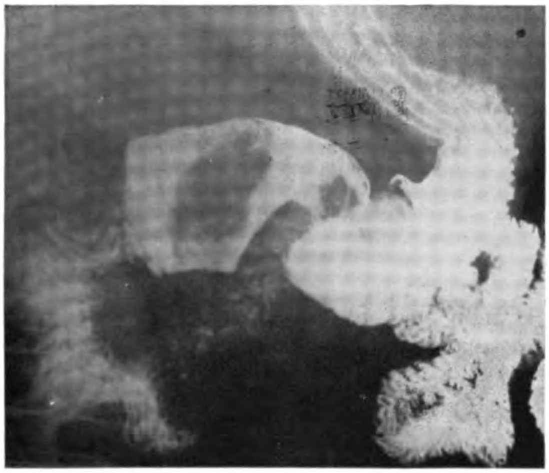

FIG. 74

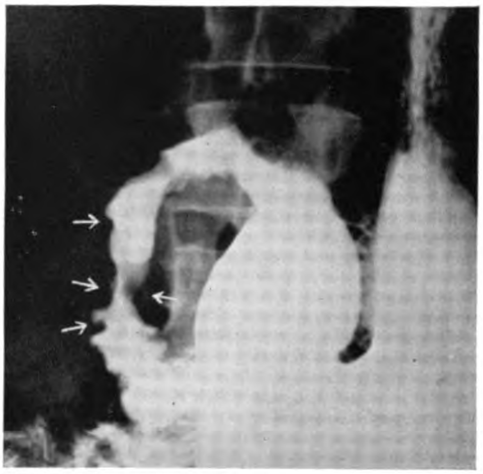

FIG. 75

FIG. 74.—Primary carcinoma of the first portion of the duodenum in a 53-year-old woman with melena, abdominal pain, anorexia and weight loss.

FIG. 75.—Infiltrative carcinoma of descending duodenum in a 47-year-old woman diagnosed clinically as peptic ulcer.

duodenum is changed to an irregularly stiffened tube. Fungating polypoid tumors project into the lumen, resulting in varying distortions. Scirrhous tumors may show nothing more than an irregular rigidity of a portion of the duodenal sweep. Ulceration is suspected when barium is deposited in craters. As a rule the duodenal sweep is not widened, but is fixed. An infrequent form of carcinoma of the duodenal bulb is one with an irregularity which cannot be differentiated from a duodenal ulcer. In these patients the clinical history is that of an ulcer, and the diagnosis is established at operation.

Lymphomas of the duodenum are relatively uncommon. They cannot be distinguished either clinically or roentgenologically from carcinoma unless the disease is part of a generalized process. A case of duodenal lymphosarcoma was reported by Poer in which the history suggested a perforated peptic ulcer. One of our verified cases was a spindle cell sarcoma which was mainly extraduodenal. A tremendous ulceration about 20 cm. in diameter had formed within the tumor mass, which was adherent to the posterior wall of the stomach, the liver and the pancreas. At necropsy this cavity was found lined with soft viable tumor tissue. Rarely discrete circular shadows due to lymphoma appear in the duodenal bulb or the descending duodenum which cannot be told apart from other lesions such as benign adenomatous polyps or heterotopic pancreatic implants. A case of duodenojejunal intussusception due to a duodenal lymphoma of this variety was reported by Knight and Black.

Leiomyomas and leiomyosarcomas of the duodenum also are infrequent, and predominate in middle aged males. Heymann and Clark, in reporting what they believed the sixteenth case of leiomyosarcoma of the duodenum, commented on the tendency of this tumor to be extraluminal if it arose from the outer muscular layer. Tumors arising from the inner coat have a tendency to bleed. In general, these tumors may be classified as intramural, submucosal, subserosal and of a dumbbell type. A characteristic of the tumor is ulceration and invasion of the adjacent organs. These tumors attain considerable size, sometimes producing pressure effects against adjacent bowel without actually invading it. As a rule, they metastasize late.

No symptoms may be noted until sufficient

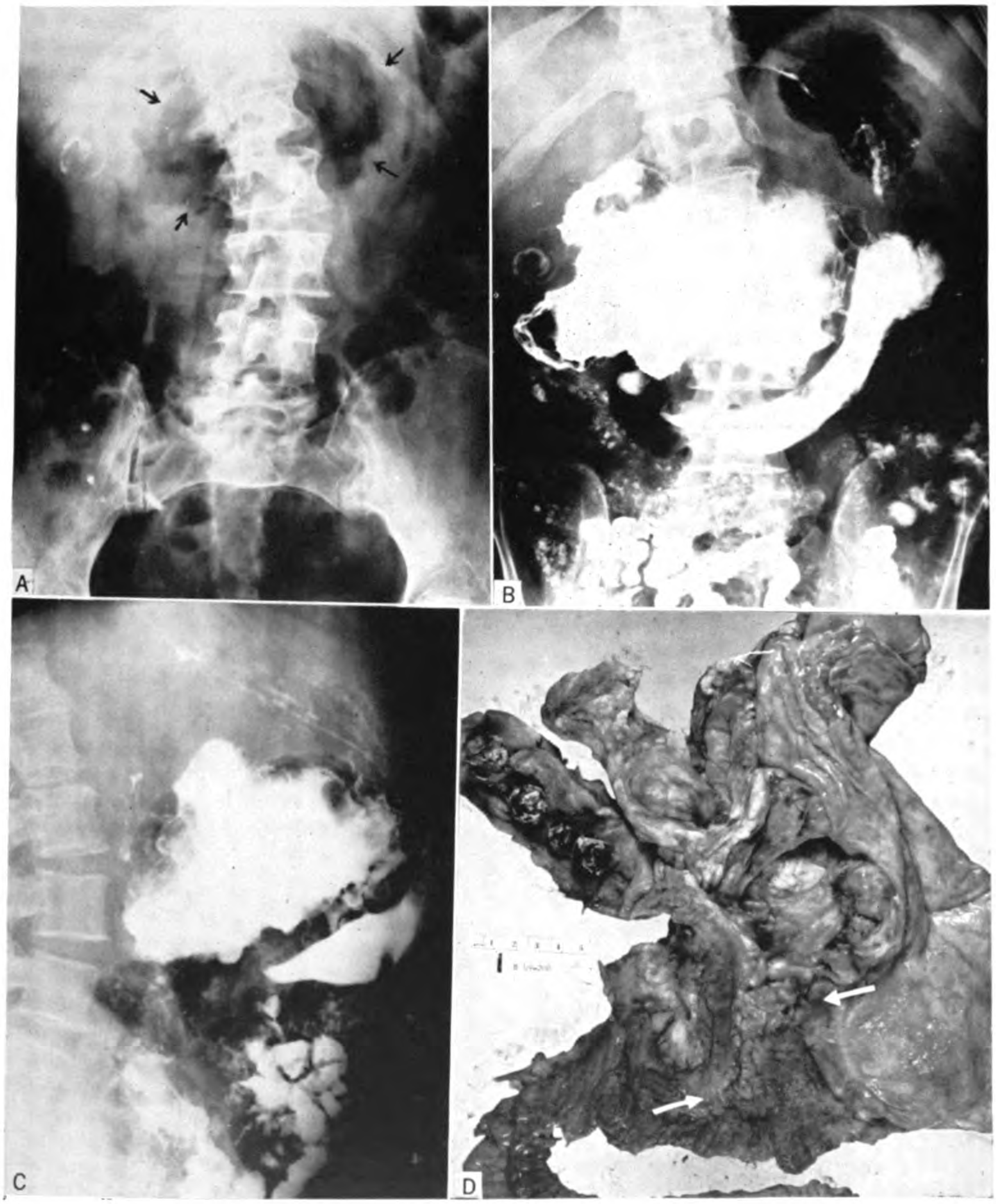

FIG. 76.—*A*, Spindle cell sarcoma in a 59-year-old female. Scout film reveals a gas containing shadow (arrows) in the epigastrium.

B, Same patient after barium meal. The barium has entered a large retroperitoneal cavity corresponding to the lesser sac.

C, Same patient, left lateral view.

D, Specimen showing tumor mass adherent to stomach, liver and pancreas. There is a large necrotic cavity in the tumor, the space being lined with soft, friable tumor tissue.

size has been reached to produce pressure changes, or ulceration of the mucous membrane occurs.

Pedunculated antral tumors can pass through the pyloric ring and rest in the duodenal bulb. These are sometimes replaceable into the stomach. Leiomyomas may do this, as indicated by the case of Child and Braunstein in which a prolapsed leiomyoma caused gastroduodenal intussusception with massive hemorrhage and severe epigastric pain.

A less frequent cause of pseudotumor in the duodenal bulb is an impacted gallstone. Occasionally a diverticulum adherent to the wall of the cap gives rise to a similar shadow.

Multiple sharply-defined circular defects or coarse granular changes may be due to hypertrophy of Brunner's glands. These are ramifying tubulo-alveolar structures in the mucosa and submucosa, abundant in the proximal half of the duodenum and disappearing towards the duodenojejunal junction. They vary in size from 0.2 to about 1 cm. in diameter. As a rule this condition is asymptomatic. However, Epstein and Cole reported a patient who had epigastric distress and vomiting. At operation the duodenal mucosa was heaped up sufficiently so as almost to obliterate its lumen, and biopsy confirmed the diagnosis of hyperplasia of Brunner's glands.

Golodner, Slobodkin and Ripstein reported papillary lymph node hyperplasia of the duodenum producing buck-shot filling defects. Such hyperplasia was considered the same as seen in the stomach, ileum and appendix and the colon and rectum. Similar radiolucent defects also were described by Nahon.

Duodenal carcinoids are infrequent, and are indistinguishable from other tumors. In a review of the literature Barnes and Young found 26 cases, 12 of which had vomiting, upper abdominal pain and jaundice, but no gastrointestinal bleeding. These authors reported 2 additional cases, 1 with repeated episodes of severe bleeding.

In differential diagnosis it is often difficult to separate duodenal cancer from those of the pancreas, the ampulla of Vater, the common bile duct, or occasionally of the gallbladder. The association of duodenal polyps with diffuse gastrointestinal polyposis is infrequent. One case observed by us had numerous colon polyps as well as a single adenomatous polyp of the duodenal bulb. Another had single pedunculated polyps in the duodenal bulb, the terminal ileum and the colon. Neither had stigmata of the Peutz-Jeghers syndrome.

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NEOPLASMS OF THE SMALL INTESTINE

Most neoplasms of the small intestine are of epithelial origin. Carcinomas occur most often in the proximal jejunum or the distal ileum as polypoid, ulcerative or sclerosing annular tumors. Combined forms are not uncommon. They begin insidiously, with initial complaints of weakness, abdominal distress and pain, occasionally diarrhea and blood in the stool. Vomiting and cramps appear when obstruction supervenes. A palpable abdominal mass appears if the tumor grows extrinsically, but usually no abdominal mass is found. With extension to adjacent viscera the type of abdominal pain may change. If the tumor penetrates retroperitoneally, back pain may be prominent. Obstructive symptoms depend on the extent and site of the block. An obstruction high in the lesser bowel will cause distressing vomiting and pain, while a block low in the ileum is more apt to cause distention and a lesser degree of vomiting. In adults, intussusception of the small intestine may be caused by malignant tumors much the same as produced by benign neoplasms or Meckel's diverticulum. Brayton and Norris, in a review of 745 cases of intussusception found that 52 per cent were of small bowel origin, and 38 per cent involved the large bowel. Benign tumors were responsible for 33 per cent and malignant growths for 21 per cent of cases.

In our 18 small intestinal neoplasms, 9 were located in the proximal jejunum, 3 in the distal jejunum, 2 in the proximal ileum and 4 were in the distal ileum. Five were annular constricting tumors which encircled and constricted the bowel. A palpable mass was present in 5, associated with extension of the growth to the adjacent viscera. In 1 the tumor involved the mesosigmoid, in another the transverse colon and in a third the greater curvature of the stomach was involved. Invasion of the mesenteric lymph nodes and the mesentery was present in 2. In 1 patient the possibility of an intra-abdominal malignancy was suggested by the presence of a Bloomer's shelf.

The radiologic diagnosis of small intestinal carcinoma requires primarily an awareness of the lesion. No gastrointestinal examination is truly "negative" until the lesser bowel has been carefully scrutinized. Radiologic manifestations are not lacking when gross obstruction exists, but the identification of a small, nonobstructive tumor may require repeated examination. Sometimes the mass itself will not be apparent, but any persistent fixation of a loop of small intestine associated with a suggestive clinical picture may be enough to warrant surgical intervention.

Small Intestinal Polyps.—Small intestinal polyps occur as single or multiple lesions. Proliferation of epithelium may cause formation of a mucosal pedicle, with polypoid proliferation on the stalk. Infrequently polyps form from lymphoid tissue within the wall of the bowel. Epithelial polyps sometimes exist as single isolated lesions, which are insignificant unless associated with bleeding or intussusception. Roberts, Peskoe and Fader noted that adenomas of the jejunum are relatively infrequent, but described 1 about 5 cm. in diameter in a 40-year-old man.

In 1929 Peutz, and in 1949 Jeghers described a form of hereditary diffuse intestinal polyposis associated with peculiar cutaneous melanin deposits on the lips, the mouth, and

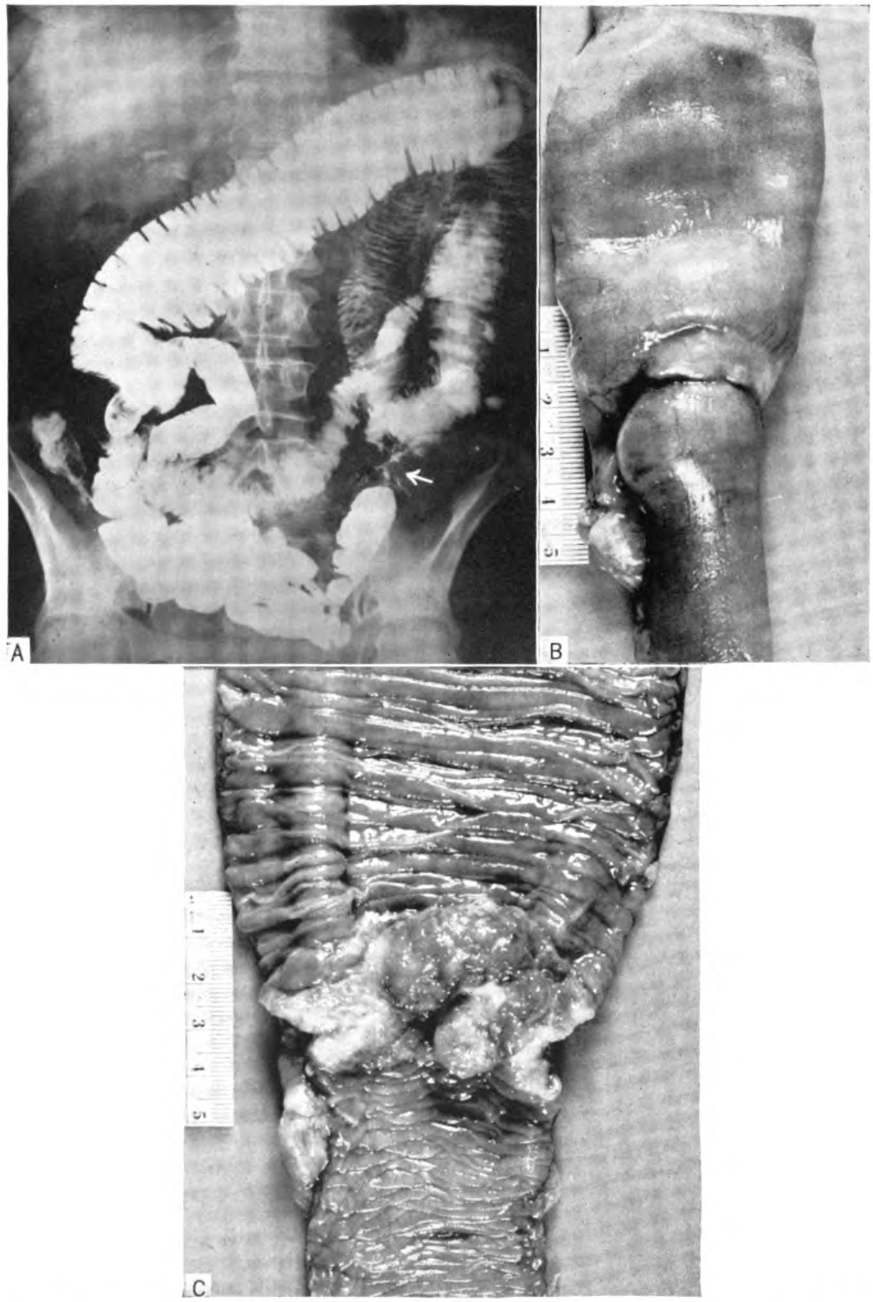

FIG. 77.—*A*, Carcinoma of the jejunum in a 52-year-old woman with recurrent attacks of abdominal pain for several months and weight loss. The jejunum is dilated, and a filling defect is present in the left lower quadrant (arrows).

B, Specimen showing dilated jejunum above ring of tumor.

C, Specimen opened.

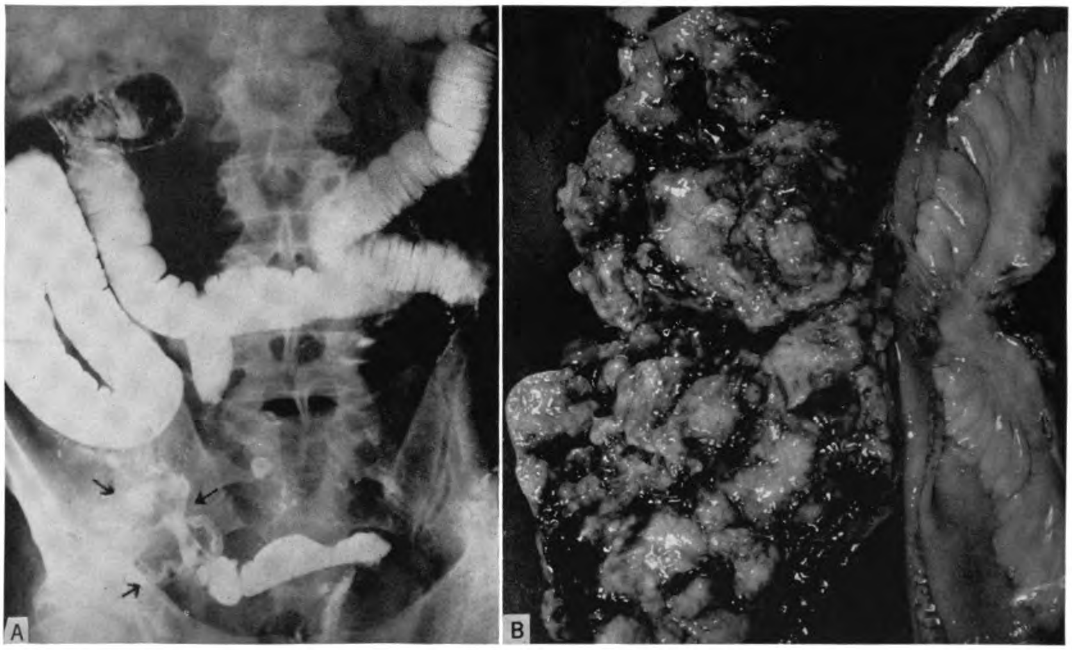

FIG. 78.—*A*, Extrinsic leiomyosarcoma of the ileum in a 60-year-old man with constipation, bloody and black stools, weakness and anemia. Lesser bowel study shows dilatation of jejunum and ileum, and irregular filling defect in distal ileum in right lower quadrant (arrows).

B, Specimen, same patient.

the extremities. These polyps are more frequent in the jejunum, but are encountered anywhere in the gastrointestinal tract including the stomach. They vary in size from about 1 to 3 cm. in diameter, and frequently are on stalks. There may be no symptoms, or the patients may complain of intermittent abdominal distress and occasionally diarrhea and melena. Intussusception is rather frequent, occurring intermittently and with variable severity. A malignant potential is attributed to these polyps. Kleitsch, Korn and Saichek reported a jejunal carcinoma in a patient with the Peutz-Jeghers syndrome in whom the presenting complaints were anemia and intestinal obstruction. In this patient colonic polyps were correctly diagnosed, but the small intestinal lesions were not identified radiologically. The radiologic diagnosis of these lesions is identical with other polyps of the gastrointestinal tract.

Mesenchymal Tumors.—Lymphomas of the small intestine are uncommon, and occur as isolated or scattered lesions, either independently or as part of a generalized process. Marcuse and Stout reported 13 cases of primary small intestinal lymphosarcoma from the Presbyterian Hospital between 1932 and 1947, and collected 179 similar cases from the literature of those years. Three of their cases and 23 from the literature survived 5 years or longer. The possibility of a lymphoma occurring as a local lesion curable by operation is noteworthy.

Lymphomas of the small intestine occur somewhat more often in the ileum than elsewhere in the small intestine. They are frequently intramural rather than intraluminal, and give rise to narrowed segments of intestine with adjacent areas of dilatation. Their presence parallels to some degree the richness of the lymphatic tissues. The disease may originate retroperitoneally, and extend to the small intestine through the mesentery, with predominantly extrinsic involvement which may become obstructive.

The clinical picture of small intestinal lymphoma is not specific. In addition to the wide variety of systemic complaints, a symptom complex very similar to sprue is produced when sufficient mucosal damage has taken place to interfere with absorption. In such patients a malabsorption syndrome predominates, with diarrhea, steatorrhea, nutritional anemia and hypoproteinemia. Sleisenger, Almy and Barr reported 4 cases of histologically proven lymphoblastoma of the small intestine and mesenteric lymph nodes, together with 13 additional cases in the literature, with all the changes of serious deficiency of intestinal absorption. Comparison with a group of 20 patients with non-tropical sprue showed that the average duration of symptoms was 69 months in the lymphoma group, as compared with 40 months in the sprue group.

Lymphomas of the small intestine in children may produce obstructive symptoms relatively early. A case of acute obstruction due to an ileal lymphosarcoma was described by Ottley in a 5-year-old child who had been operated upon because of symptoms of acute appendicitis. This patient had a recurrence 18 months later, and expired about 3 years after the first operation. In one of our young patients a resected loop of jejunum was variously diagnosed as regional enteritis, nonspecific granuloma and reticulum cell sarcoma by several pathologists. The lesion recurred 4 years later, and at necropsy a lymphoma originating in the root of the mesentery was found extending to the small intestine, fixing several of the loops and causing obstruction. Cutler, Stark and Scott remarked that intestinal tract neoplasms in infants and children should be considered lymphoma until proved otherwise.

The radiologic appearance of small intestinal lymphoma is inconstant. In some patients with diffuse involvement, the picture of sprue, or less often, that of a nonstenosing regional enteritis may be simulated. With small tumors the picture may be one of intestinal obstruction or intussusception. The presence of definitely and extensively stenosing lesions is uncommon, as is extension to the cecum. Localized areas of dilatation, alteration of mucosal patterns, diminished peristalsis, rigidity of small intestinal loops, fixation, and changes in the passage rate of barium have all been observed. The appearance of fistulas is uncommon, but we have observed 2 such cases with entero-enteric passageways.

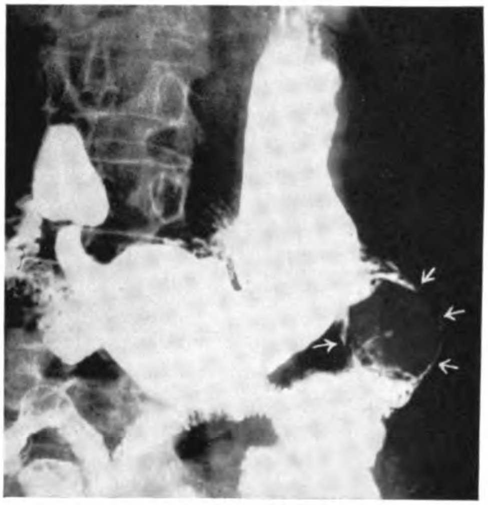

FIG. 79.—Jejunal leiomyoma in a 56-year-old woman with abdominal and back pain, probably unrelated to the lesion.

Leiomyomas and leiomyosarcomas of the small intestine are infrequent tumors, occurring anywhere in the small intestine, including Meckel's diverticulum. They cannot be distinguished radiologically. These new growths occur in a predominantly extraluminal form, with the bulk of the tumor extending into the peritoneal cavity producing only slight narrowing of the intestinal lumen and moderate mucosal changes. These may be large enough to be easily palpated, and when bulky may break down forming large areas of central necrosis and fistulas. Abscess formation may be superimposed by infection. The intraluminal type of leiomyoma or leiomyosarcoma has its major portion within the intestinal lumen. The tumor often is submucosal, and produces rather marked alterations of the overlying mucosa. These may be symptomless growths, unless they become sufficiently large to obstruct or initiate intussusception. As a rule, these do not become as large as the extraluminal variety. A third, dumbbell variety is created when an intraluminal growth perforates the wall of the bowel and extends as a combined intraluminal and extraluminal tumor. With ulceration of the overlying mucosa, bleeding and pain simulating duodenal ulcer may appear. Perforation of the tumor can cause peritonitis.

A relatively uncommon tumor of the small intestine seen in middle life is the lipoma. This usually is a smoothly outlined sessile or pedunculated tumor, but occasionally occurs in a lobulated form. Unless obstructive, no symptoms appear. Bleeding is associated with ulceration of the lipoma, or with intussusception.

Hemangiomas of the small intestine also are relatively uncommon benign tumors, rarely diagnosed preoperatively or during life. Occasionally they cause intussusception. The presence of skin or mucous membrane hemangiomas sometimes suggests the presence of a similar lesion of the lesser bowel if intestinal obstruction occurs. These tumors are characterized by multiple cavernously dilated venous structures. Usually they are quite small, but may be of fair size and occasionally are diffusely infiltrating or multiple. Narrowing of the lumen of the bowel is observed with the infiltrating form of hemangioma. Polypoid hemangiomas appear as circumscribed lesions. The presence of closely clustered or layered phleboliths may help identify an intestinal hemangioma if these stones can be identified within or immediately proximal to the intestine.

There is a tendency for melanomas to metastasize to the small intestine. Beirne noted that of 33 melanomas reported 22 were metastatic. He reported a case of an apparently primary melanoma of the intestine in a 39-year-old male who had been treated for gastrointestinal bleeding, presumably from an ulcer. Weight loss, anemia, cramps and tarry stools were the presenting complaints. Radiologic examination disclosed multiple filling defects in the jejunum and ileum, as well as a deformity of the descending limb of the duodenum. These were diagnosed as multiple polyps, but at operation the mucosa was found studded with dozens of firm polypoid malignant melanomas of all sizes and shapes, ulcerative and smooth, varying in color from pink to jet black.

Another rare cause for small intraluminal polypoid masses is Kaposi's disease. This usually is manifested by multiple cutaneous idiopathic hemorrhagic sarcomas, and may be associated with similar lesions in the small intestine. A case in which the roentgenograms revealed multiple small intestinal polyps of this origin as a cause of gastrointestinal bleeding was reported by Boone and Kratzman, and another was reported by Seagrave. Evidence that this tumor may be primary in the viscera without skin involvement was presented by Aegerter and Peale.

Carcinoid tumors arise from specialized granular epithelial cells of the crypts of Lieberkuhn. The yellow cells of Kultschitzky occur most frequently in the appendix, but also are frequent in the lesser bowel, particularly the ileum. In a review of 356 carcinoids of the gastrointestinal tract, MacDonald noted that 149 were extra-appendiceal. Of these, 74 were in the ileum, 17 in the jejunum, 14 in the stomach, 13 in the rectum and 8 in the duodenum. These tumors usually are covered by intact mucosa, and are nodular. Ulceration of the mucosa may occur. The tumor extends into the muscularis and serosa. Peritoneal spread is accompanied by fibrotic contraction, kinking of the intestine and consequent obstruction, which frequently is the presenting symptom.

These tumors once were considered relatively benign, but it has become apparent that they have a definite malignant potential, even though some of the usual standards for malignancy such as mitoses and cell anaplasia are not entirely applicable to carcinoids. MacDonald noted that the majority of patients with metastases to lymph nodes and other organs rarely had evidences of mitoses and anaplasia. Because of this the degree of malignancy must be determined on the basis of gross and microscopic invasiveness. The tumor first invades the muscle coats, reaching the lymph nodes by the lymphatics and passing to the liver through the circulation. Recently Thorson, Biorck, Bjorkman and Waldenstrom, and Bean, Olch and Weinberg have supplied evidence that a relationship exists between widely metastasized carcinoids and certain bizarre clinical alterations such as telangiectasis and a proliferative and thrombotic disorganization of blood vessels of the skin with transient flushing, mottled cyanosis, diarrhea, asthma and signs of valvular lesions of the right heart and congestive failure. MacDonald also noted a concomitance of gastric and duodenal ulcers with carcinoid. In the 21 cases he reported there were 8 which also had peptic ulcers.

The radiologic manifestations of carcinoid of the small intestine are not specific. The distention of the bowel seen with obstruction does not indicate the underlying cause. In some patients with infiltration of the muscularis there may be tubulation of the small intestine simulating regional enteritis. In the unobstructive phase a suggestion as to the presence of carcinoid may be warranted when loops of small intestine are relatively fixed in position and persist in a coiled pattern. Transient small intestinal obstruction with dilatation of the proximal ileum and jejunum also may point towards a partial obstruction. The presence of systemic changes of flushing, cutaneous disorders and right heart disease may likewise point towards the necessity of ruling out carcinoid.

Fibromas of the small intestine are infrequent tumors, which are usually undiagnosed unless bleeding or intussusception occur. These tumors occur within the wall of the bowel, and project into the lumen as relatively circumscribed smooth-walled structures. They may become polypoid, and as such have a greater tendency to produce intussusception. In one of our patients a barium enema was done because of abdominal pain with melena. The terminal ileal loops filled, and a circumscribed lesion was identified in the midileum. This was identified at operation as an ileal fibroma.

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TUMORS OF THE APPENDIX

Most appendiceal tumors are carcinoids. In contrast to those elsewhere in the intestinal tract, these are less often malignant, and frequently are identified incidentally at necropsy. Occasionally they produce symptoms suggestive of acute appendicitis. The true nature of the lesion is then identified on histologic examination of the removed appendix.

Parkhurst and Cassidy mentioned that carcinoids constitute about 90 per cent of appendiceal tumors. About 8 per cent of the remainder are mucoceles, resulting from obstruction at the base of the appendix. Usually these are quite small, but they do reach sufficient size to be easily felt through the abdomen. The mucoid content may be the starting point for pseudomyxoma peritonei, which occurs when the cyst ruptures, spreading its contents in the peritoneal cavity. Large masses of mucinoid cystic lesions then appear which continue to grow. The diagnosis of a mucocele sometimes is suggested on x-ray examination by a mass impinging against the base of the cecum. Intussusception is an infrequent complication of mucoceles.

Carcinomas of the appendix are relatively uncommon, their incidence being about 3 per cent. Niceberg, Feldman and Mandelberg observed that appendiceal adenocarcinoma has never been diagnosed preoperatively, the symptoms being those of acute appendicitis. They collected 49 cases, of which 31 were males. The ages varied from 26 to 79 years, with a peak incidence in the fifth to seventh decades. These carcinomas behave like ordinary colonic carcinomas, spreading to the lymphatics or metastasizing hematogenously. Production of symptoms is indicative of appendiceal obstruction by tumor infiltration, or perforation with abdominal spread.

Occasionally an appendiceal carcinoma becomes mucoid-producing. We saw such a case in a 27-year-old male with extensive

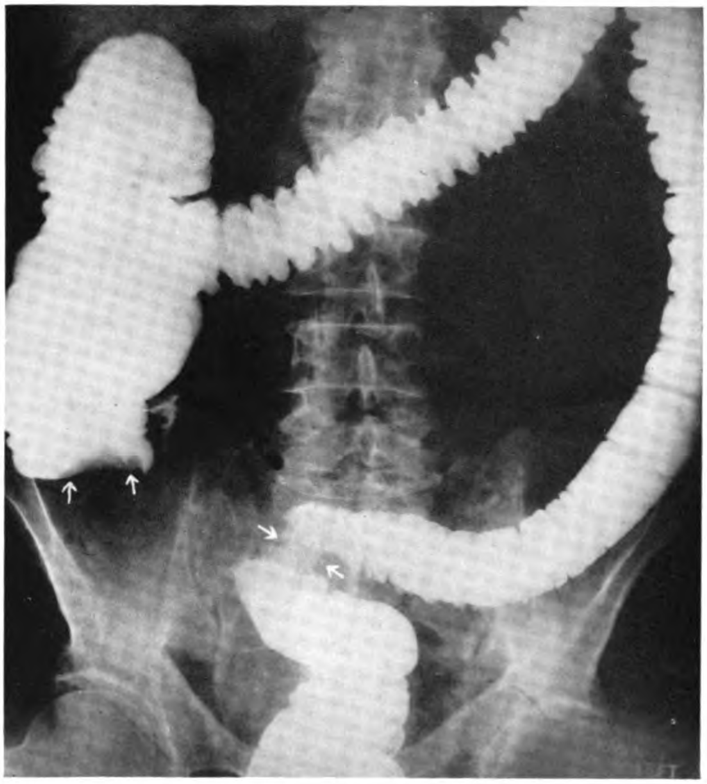

FIG. 80.—Lymphosarcoma of the appendix in a 65-year-old man with massive rectal bleeding. A large mass was palpated in the right lower quadrant, extending into the pelvis. Barium enema film reveals arcuate defect in caput cecum, and annular deformity at the rectosigmoid (arrows). Examination of operative specimen revealed a stubby appendix about 1.5 cm. in diameter surrounded by a large mass extending into the pelvis.

ascites. At operation there were intra-abdominal metastases from a verified mucoid carcinoma of the appendix. Another carcinoma of the appendix verified at necropsy extended to the peritoneal cavity and formed a Krukenberg tumor in the ovary of a 51-year-old woman. On barium enema examination the appendix filled, its distal end being dilated and irregular. This was due to perforation of a carcinoma in the distal end of the appendix.

Lymphoma of the appendix is quite rare. One patient 65 years old was admitted with extensive bleeding attributed to a sigmoid polyp. He also had a palpable lower abdominal mass. Radiographic examination disclosed diffuse distention of the small and large bowel. A bleeding polyp was seen sigmoidoscopically at 18 cm., but 4 cm. beyond this a gritty, scirrhous growth was present. A barium enema examination revealed a defect in the sigmoid, together with irregularity of the cecum and upward displacement of the ileum. At operation a large mass adherent to the bladder was found, filling the pelvis. This was affixed to the sigmoid, and it was evident that it originated from the appendix, which was a stubby stalk about 1.5 cm. in diameter between the mass and the cecum. Liver metastases were present, together with large lymph nodes in the mesentery. Histologic examination was reported as lymphosarcoma.

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LARGE INTESTINAL TUMORS

Most malignant tumors of the colon are adenocarcinomas, and occasionally scirrhous and mucoid carcinomas are encountered. There is a tendency for these tumors to originate in epithelial polyps, but the radiologist cannot distinguish a benign from a malignant polyp with any degree of certainty. The identification of a colonic malignancy as early as possible is vastly important because some are curable. The identification of a single polyp suggests rather than excludes the possibility of multiple lesions. There is a very poor arithmetic correlation between the number of polyps seen radiologically with those found at necropsy. Accuracy at the operating table falls midway between these extremes.

Carcinomas of the colon, among the most frequent tumors seen in adults, have a predilection for the sigmoid and rectum. Fully three-quarters of them occur in this region. Next in frequence of location are the ascending colon, the transverse colon, the splenic flexure, the cecum and the descending colon. These are mainly tumors of middle life. However, colonic neoplasms are seen in relatively young people, including children. Williams reported that none of the 53 children he gathered from the literature lived more than 4 years after the diagnosis had been made. He stressed the fact that symptoms were non-specific, with pain, nausea and vomiting being the common complaints. In children the usual diagnosis was acute appendicitis, or sometimes tuberculous peritonitis.

A relatively infrequent form of colonic carcinoma is the linitis plastica type, which simulates phlegmonous inflammation because of a coexistent subacute or chronic inflammation, of a relatively long segment of bowel. In reporting 4 such cases, Laufman and Saphir commented that the underlying malignant nature of the lesion had not been recognized clinically.

Radiologically, neoplasms of the colon can be classified on the basis of their gross structure as proliferative, with cauliflower-like projections into the bowel, as polypoid, or as annular lesions encircling the bowel partly or completely. Ulcerative neoplasms burrow into the mucosa and submucosa, producing a fibrotic inflammatory reaction. Invasion of the bowel wall is followed by extension of the tumor into regional lymph nodes by way of the lymphatics and further spread systemically through the circulation. The size of a tumor bears no constant relationship to its metastases, so that a tumor almost unidentifiable grossly can produce large deposits in the regional lymph nodes, the liver, the mesentery and omentum, the peritoneal cavity and the retroperitoneal areas. Tumor implants against the serosa of adjacent stomach and bowel is not uncommon, and sometimes simulates multiple tumors. Fistula formation to the gallbladder, the small intestine, the urinary bladder and the retroperitoneal structures all have been observed. Infrequently tumors invade the sacrum and lumbar spine, with initial symptoms referable to the cauda equina.

Malignant change associated with ulcerative colitis is noteworthy. Cattell and Buehme reported that the average interval from the onset of colitis to the development of cancer was approximately 9 years. Such degeneration usually is highly malignant, and associated with early metastases. These new growths are more frequent in younger people. Johnson and Orr noted that in 40 cases from the literature the average age at which carcinoma was discovered was 40 years, while in patients without colitis the average age was 53 years.

Radiologic examination of the colon usually is initiated by suspicion of some disorder, often on the basis of an educated guess. A high index of curiosity often results in the identification of lesions which otherwise would have been overlooked. It is imperative that the bowel be thoroughly cleansed, but care should be taken not to overprepare the patient with harsh cathartics and overenthusiastic enemas. This may result in atonicity of the bowel, a condition which also is observed when antispasmodics or anticholinergic drugs have been used for some time before the colon examination. Suffice it to say that while preparation of the patient may be standardized, the response of the individual is by no means uniform. This is especially true of young children and elderly patients. Nevertheless, the importance of a properly prepared bowel in the investigation of possible polyps or nonobstructive lesions cannot be overemphasized. We are among those who use from 2 to 3 teaspoonfuls of powdered tannic acid in our enema mixtures.

The flow of barium is controlled by the fluoroscopist, whose responsibility it is to see that all parts of the bowel are carefully scrutinized. Appropriate spot films are made throughout the procedure, followed by additional films after the colon is filled, after evacuation and after the instillation of air. We have no standardized set for colon examinations, utilizing whatever projections are necessary for bringing out a pathologic change. All films are scrutinized while the patient is in the department, it being the responsibility of the fluoroscopist to determine when the study is complete. Even under the best of circumstances there are times when an examination is inadequate. There should be no hesitation in requesting a restudy. The consequences of overlooking a lesion which is potentially curable places a great responsibility upon the radiologist. It is far better to re-examine the patient than to trust to luck that a small defect is not of clinical importance. Indeed, we feel that the most difficult diagnosis to make is that of "negative colon examination."

Tumors of the Proximal Third of the Colon.—For present purposes this area is defined as the cecum, ascending colon and the hepatic flexure approximately up to its junction with the transverse colon. The clinical picture with tumors of this area are deceptive. Not infrequently the only indication of such a cancer are vague symptoms of weakness, anemia, nausea and weight loss. With infection and perforation pain, fever, chills and diarrhea all are encountered. While these lesions aften bleed, gross hemorrhage is uncommon, occurring but 3 times in a series of 66 patients.

Tumors higher in the ascending colon are even more difficult to identify clinically. Until these lesions are large enough to obstruct the bowel, they may be entirely silent, or present a wide variety of nonspecific symptoms and occult bleeding. When obstruction supervenes, its onset usually is insidious, with mild abdominal distress later followed by pain, cramps and vomiting. If only partial block is produced by a cecal neoplasm, the patient may have relatively little pain, but frequently complains of progressive distention which may reach huge proportions. In several of our patients the small intestine was dilated up to 6 or 7 cm. in diameter, and at operation several liters of fluid stool were aspirated before the bowel collapsed. More often distention is less pronounced, and the intestinal pattern is quite varied. Occasionally the jejunum is distended more than the ileum. At other times the possibility of obstruction is suggested by distended loops of small intestine in the right lower quadrant. This often is intermittent and variable, and when associated with distention of the colon may be quite difficult to interpret.

Obstructions in the ascending colon are followed by slow, progressive and ultimately extensive dilatation of the cecum. Lowman and Davis considered a cecum over 9 cm. in diameter as one in danger of perforation. When the cecum does perforate a tremendous pneumoperitoneum may appear, and the outlook of the patient is indeed bad.

A review of 40 verified cases of cecal carcinoma showed that no obstruction was pres-

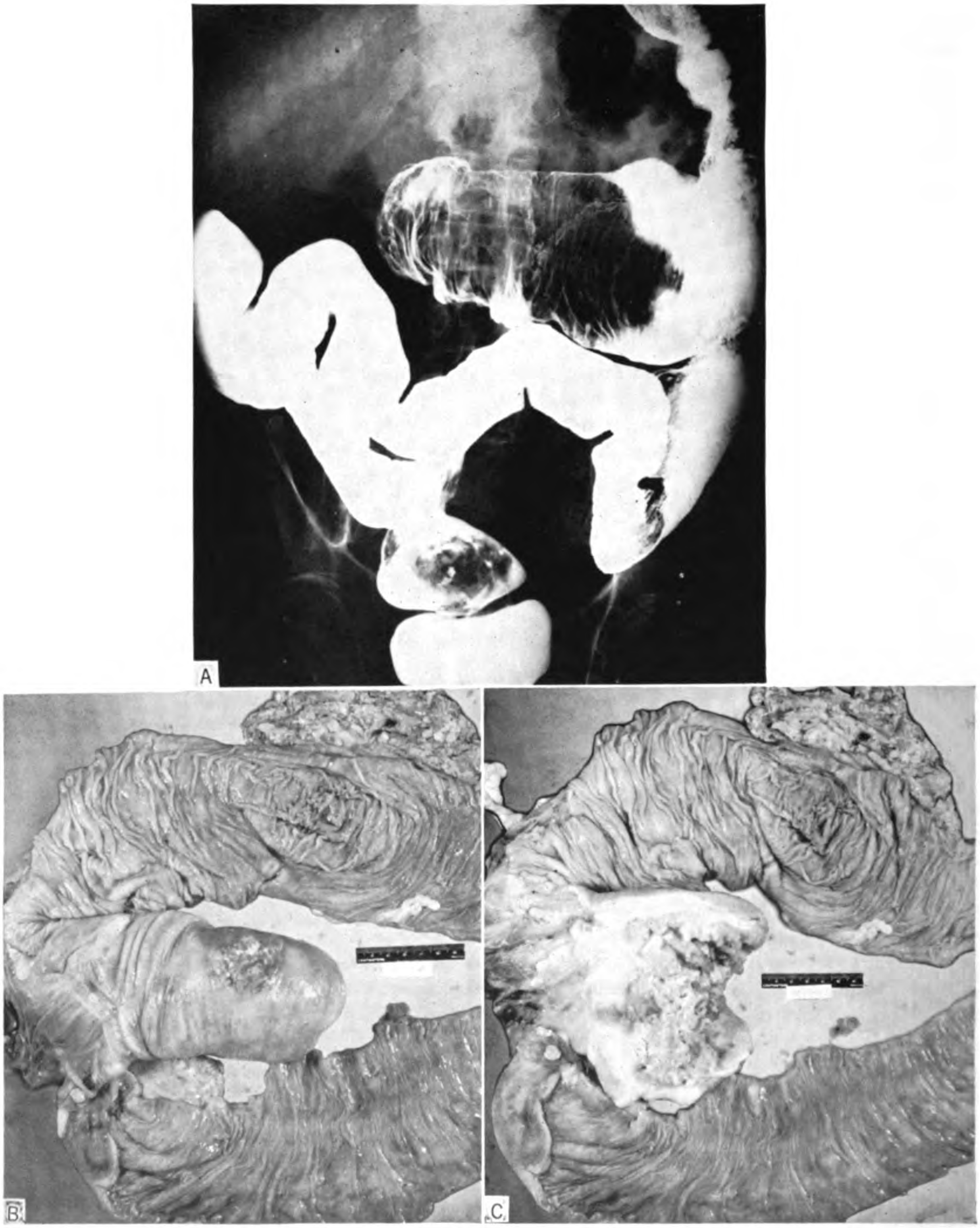

Fig. 81.—*A*, Carcinoma of the cecum intussuscepting into transverse colon in a 70-year-old man. Note the dilated bowel with coiled-spring appearance of the intussusceptum and intussuscepiens.

B, Operative specimen, closed. The intussusception was irreducible.

C, Same patient, open specimen revealing adenocarcinoma.

ent in 21. Four had mild obstruction as reflected by moderate ileal distention. In 5, chronic recurrent ileocecocolic intussusception was present. In 10, advanced obstruction was reflected by severe lesser bowel dilatation. Barium enema examination and examination from above did not portray the tumor equally well. The most difficult ones to identify were the non-obstructing, relatively flat infiltrating tumors. These were seen more often distal to the ileocecal valve, and caused little disturbance in the flow of intestinal contents. Larger tumors protruding intraluminally or encircling the bowel could be demonstrated radiologically with more certainty, and were associated with variable lesser bowel distention and symptoms of intestinal obstruction. The hazards of relying on examination of the colon as part of an upper gastrointestinal examination was exemplified by 10 cases in which the latter examination had been reported as normal, while barium enema clearly delineated the tumor. This occurred in 7 cases of cecal tumor and 3 of the ascending colon, and was attributed to the free passage of relatively liquid bowel contents through a bowel which was still quite patent. However, when examined by an opaque enema, a ball-valve-like action appears which obstructs the flow of contrast material, and often reveals an abnormal appearance of the wall of the bowel.

Intussusception, often of a chronic recurrent variety, was accompanied by the classical picture of invagination of the bowel headed by the neoplasm, with concentric rings about the intussusepiens. In 3 of our cases this could be easily reduced by hydrostatic pressure, and the polypoid nature of the underlying lesion was clear on post-evacuation and air contrast examinations.

Perforation of the colon occurred in 9 patients with cecal tumors. In these, a mass was present in 5, a diagnosis of pericecal abscess being made. Direct extension of the neoplasm to the ileum was infrequent. In 3 the tumor involved the serosal coat of the terminal ileal coil immediately adjacent to the caput cecum. Pneumoperitoneum occurred in 2 cases, both with dramatic pictures of shock and large quantities of free gas in the abdomen.

There were 4 cases in which the diagnosis was missed, but made on re-examination within 3 weeks thereafter. How many cases were missed, and identified by someone else unknown to us is something which we cannot know, but the possibility exists constantly when an examination is reported as normal.

In 26 cases of carcinomas of the ascending colon and hepatic flexure the clinical picture was similar to that of cecal carcinoma. However, the tendency to obstruction was somewhat less. There were 15 cases with no obstructive symptoms, and in 5 only mild obstruction was present. However, 6 had advanced obstruction with pronounced dilatation of the cecum and lesser bowel. Rupture of distended bowel proximal to the obstruction occurred in 3. In 2 of these we were able to identify fruits pit in the markedly dilated gas and fluid filled cecum. At operation chicken and fish bones were found as well.

Tumors of the ascending colon and hepatic flexure perforate, forming fistulæ, as occurred in 5 of our cases. One presented retroperitoneally and established a draining sinus communicating with the perirenal area. Injection of this tract showed entrance of contrast medium into the hepatic flexure. In another the fistula lead to the inferior surface of the liver, while in the remaining 3 cases the tracts led to the duodenum and gallbladder.

An interesting change in patients with ascending colon tumors was the wide variation in dilatation of the proximal colon and cecum present from time to time. At times a distended, tympanitic cecum was easily felt, while a few hours later this disappeared. Nothing more than moderate pain was associated with this transient distention.

The association of polyps with carcinoma of the proximal third of the colon must be mentioned. In 6 cases benign adenomatous polyps were seen close to the tumor, strongly indicating that the cancer originated from a similar lesion. Intussusception was infrequent in the ascending colon tumor group, occurring once in the present series.

Carcinomas of the Transverse Colon.—In this section are included tumors involving the colon from the distal hepatic flexure to the distal portion of the splenic flexure. Forty consecutive verified cases were re-

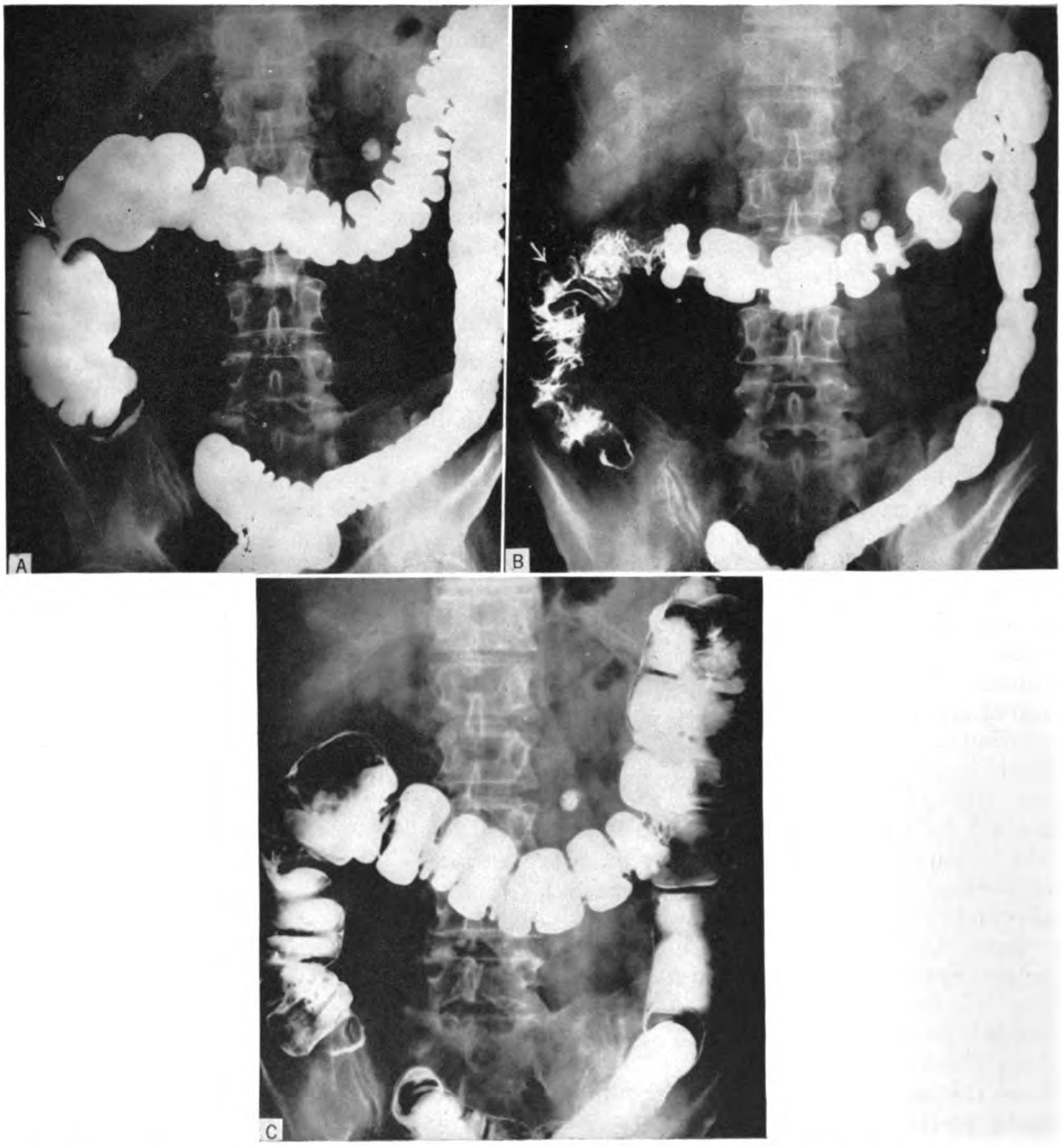

FIG. 82.—*A*, Annular carcinoma of proximal limb of the hepatic flexure in a 57-year-old man with secondary anemia, weakness and weight loss. A slight delay was noted at the site of the lesion (arrows).

B, Post-evacuation study, same patient. The mucosal pattern is but slightly altered at the site of the lesion (arrows).

C, Air contrast examination, same patient. The annular deformity is hidden partly by the adjacent distensible bowel.

viewed as background. The clinical picture is one of vague abdominal symptoms, weight loss, easy fatigability, unexplained anemia, and occasional nausea, vomiting and cramps. Patients in the middle age group were affected most often, but in our series there were 3 patients between 30 and 40 years old. Some patients noted that abdominal pain and cramps were aggravated by cathartics. Nausea and vomiting occurred as obstruction increased, when relatively constant pain also was noted. The presence of a mass on physical examination sometimes pointed to the correct diagnosis, but this was found only

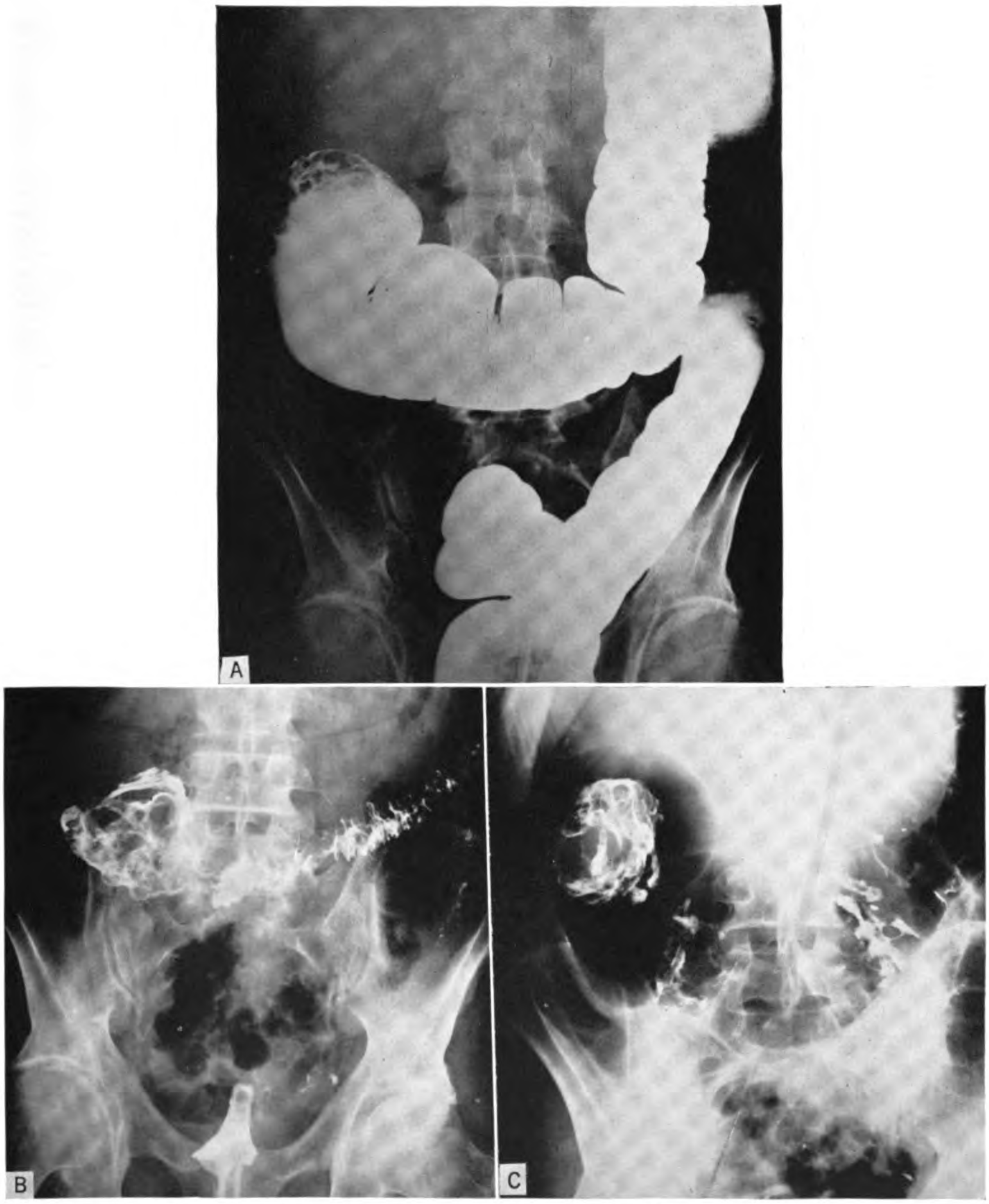

Fig. 83.—*A*, Polypoid carcinoma of hepatic flexure in a 70-year-old man acting as a one way valve, permitting fecal current to flow properly but obstructing a barium enema.

B, Same patient, post-evacuation roentgenogram. The bowel has collapsed about the tumor.

C, Same patient, air contrast study. The tumor is clearly outlined by air (arrows).

in bulky tumors. Occasionally bright red blood was seen in the stool. Three patients had tarry stools. Occasionally diarrhea was present. A variable degree of intermittent distention of the ascending colon and cecum was seen incident to partial obstruction, often with spontaneous relief. Acute symptoms were consequent to obstruction and to necrosis of the tumor. Perforation and fistulæ were relatively infrequent. Perforation was observed in 5 patients. In 1 the initial symptoms were due to a subphrenic abscess,

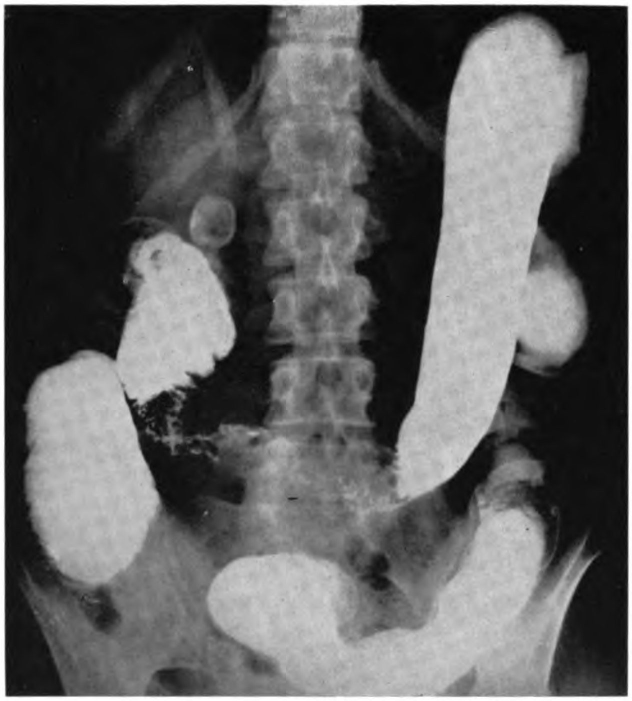

FIG. 84.—Carcinoma of transverse colon 14 cm. long in a 52-year-old woman with a palpable mid-abdominal mass.

the transverse colon carcinoma being relatively asymptomatic and identified on barium enema examination. In 3, gastrocolic or colo-duodeno-enteric fistulas were formed. In 1 case perforation of a necrotic tumor produced a pronounced inflammatory mass around the transverse colon.

From the symptomatic viewpoint, obstruction of the fecal current is quite important. Incomplete obstruction can exist with no alarming symptoms. The colon proximal to the tumor may dilate intermittently, with moderate and variable dilatation of the small intestine. In the present group 29 showed no clinical evidence of obstruction. Of the remaining 11, 8 had acute intestinal obstruction with distention of the ascending colon and of the lesser bowel up to the proximal jejunum. Vomiting was rather late.

Barium enema examination revealed obstruction in 18 patients. Volvulus of the cecum as a complication of obstruction occurred twice, with the cecum high in the left upper abdomen. Recurrent colocolic intussusception occurred in 2 patients. Extension of the tumor with involvement of the greater curvature of the stomach occurred in 3 patients, and the spleen capsule was infiltrated in another. Extension of tumor to the serosa of the adjacent ileum caused acute small bowel obstruction in 2 cases. Eight had dilatation of the cecum exceeding 9 cm. in diameter. However, actual perforation of the cecum was not observed in this group. In all the dilatation of the cecum was accompanied by pronounced distention of the lesser bowel, particularly the ileum, which may have acted as a safety valve. A competent ileocecal valve may result in marked dilatation of the colon with relatively little lesser bowel distention.

Multiple tumors were present in 3 patients in this series. One had a double neoplasm and another had three separate carcinomas of the transverse colon, together with scattered adenomatous polyps. The third patient had tumors of the rectosigmoid and rectum as well as a carcinoma of the transverse colon.

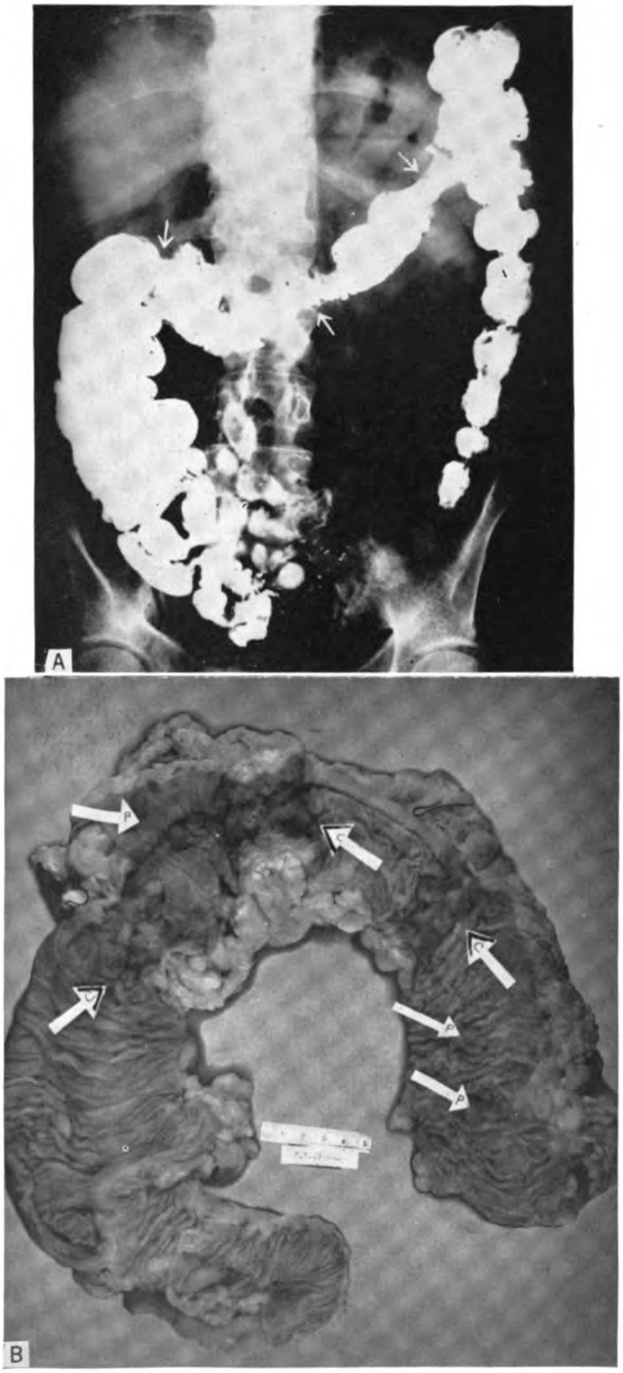

FIG. 85.—*A*, Three carcinomas of transverse colon originating in polyps in a 68-year-old man with anemia and weight loss (arrows).

B, Specimen. The arrows outlined in black indicate the carcinomas, and the others point to polyps.

Transverse colon tumors are identified radiologically by defects which vary from a complete obstruction to a small intraluminal protrusion or rigidity of the bowel wall. Tumors which encircle the lumen produce an appearance like that of a mushroom seen on end, the stalk corresponding to the lumen. When the contrast material passes through the narrowed area readily, a somewhat curvilinear convex rigidity is visible at the proximal and distal ends of the tumor. Cauliflower and fungating tumors on one wall appear as broad sessile polyps, which sometimes achieve sufficient bulk to block the intestine. It has often been said that neoplasms of the bowel are relatively short. Nevertheless, in this series tumors which were from 14 to 18 cm. long were seen 4 times. Infrequently, spasm of Cannon's sphincter in the proximal transverse colon may simulate an intrinsic lesion. If this is suspected, the examination should be repeated.

The same danger of missing lesions of the transverse colon on upper gastrointestinal examination appeared as with proximal bowel tumors. There were 5 cases in which gastrointestinal examinations followed for 24 hours showed no indication of a transverse colon tumor, while barium enema examinations disclosed definite lesions. There were 2 patients in whom the diagnosis of transverse colon tumor was made at operation, undertaken because of a diagnosis of carcinoma of the stomach. Surgery disclosed tumors originating in the transverse colon, fixed to the gastric serosa and extending through the mucularis to the mucosa. In another case a transverse colon tumor was found to be due to extension from an undiagnosed gastric carcinoma.

The danger of prolonged decompression of a dilated small intestine due to obstruction of the transverse colon was exemplified by 3 patients. All had marked improvement after introduction of a decompressive tube. Two expired after what we believe was an unwise delay in surgery. In both, advanced ulcerations were found in the ascending colon and in the lesser bowel. The mistake in diagnosis had been overlooking a transverse colon lesion by failing to do a barium enema examination in patients diagnosed as having lesser bowel obstruction, without accurately determining the cause.

Carcinoma of the Distal Third of the Colon.—Included here are tumors of the descending, the sigmoid and the anorectal colon. These new growths are most frequent in the sigmoid, with the anorectal area next in order. Of 110 cases reviewed, 65 were in the sigmoid, 30 in the anorectal region, and 15 in the descending bowel. The histologic types and growth patterns were much the same as elsewhere in the colon. Thirteen were over 10 cm. long. Concomitant polyposis was observed in 15.

The clinical picture in most cases was dominated by rectal bleeding. Changes in bowel habits, abdominal pain, intermittent fever, easy fatigability, anorexia, anemia and weight loss were prominent systemic manifestations. Pain on defecation and tenesmus was seen rather often with low bowel tumors. Melena was infrequent. The presence of a mass on physical examination was limited to those with bulky tumors.

The occurrence of intestinal obstruction was particularly interesting. In the cases of sigmoid carcinoma reviewed, there were 10 with acute intestinal obstruction as reflected by pronounced lesser bowel and colon distention, with abdominal distress and vomiting. Five others had less severe obstructive symptoms, with no specific pattern of gaseous distention. No obstruction could be diagnosed in the remaining 50 patients. Obstruction was seen in 3 patients with anorectal tumors, and in 10 of the 30 patients with descending colon tumors. It was not infrequent for patients with partial or almost complete obstruction on barium enema study to be relatively asymptomatic. Intermittency of symptoms was noteworthy, this being caused by temporary obstruction possibly by impaction of stool. In several patients sudden onset of obstruction followed operation for other abdominal disorders such as fibroids or ovarian cysts.

Perforation was less common than in the ascending and transverse colon tumors. In the group of sigmoid neoplasms, there were 8 which had perforated. In 1 case the tumor extended directly into the sacrum, resulting in a clinical picture of a cauda equina tumor. The colon neoplasm was not recognized until

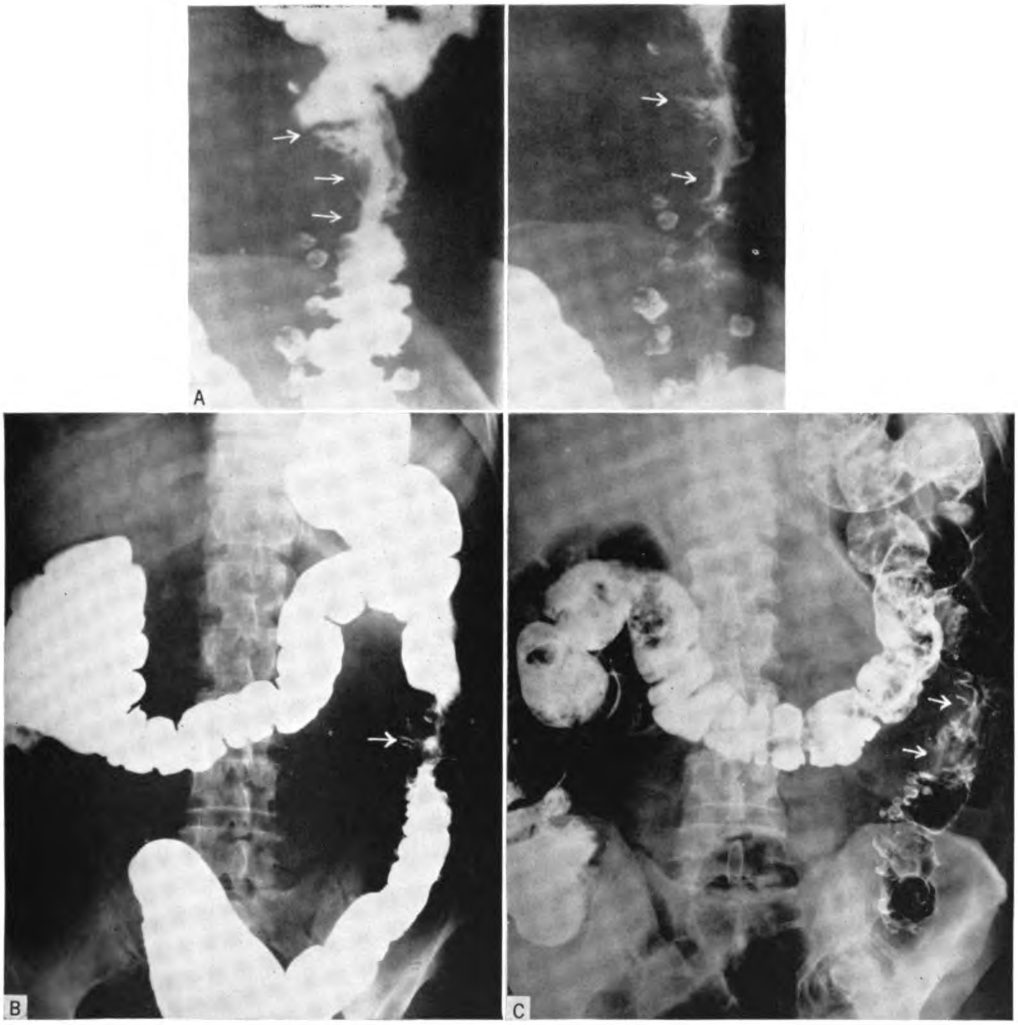

FIG. 86.—*A*, Adenocarcinoma of descending colon in a 62-year-old woman with diverticulitis. Spot film made while enema was flowing discloses scattered diverticulæ and a defect in the medial aspect of the bowel wall (arrows). The next frame was made a few seconds later during a moment of spasm of the bowel.

B, Same patient, enema film. Note the defect in the descending colon. Some linear streaks of barium are seen in its middle (arrows).

C, Same patient, air contrast study. The rigidity of the medial wall of the descending colon is again visible (arrows).

laminectomy for a tumor compressing the distal end of the caudal sac disclosed adenocarcinoma, and barium enema thereafter revealed a sessile tumor in contact with the sacrum. In the other cases the tumor invaded the adjacent viscera. In 3 cases fistulæ formed between the vagina and the sigmoid, and in 1 a rectovesical fistula appeared. In 2 cases tumors of the descending colon perforated and caused pericolonic collections. In 3 cases perforations into the soft tissues about the rectum occurred, and in 1 a sinus tract draining to the perirectal area developed.

Diverticula were noted frequently together with sigmoid carcinomas. In a few these obscured the concomitant neoplasm. No relationship could be established between these

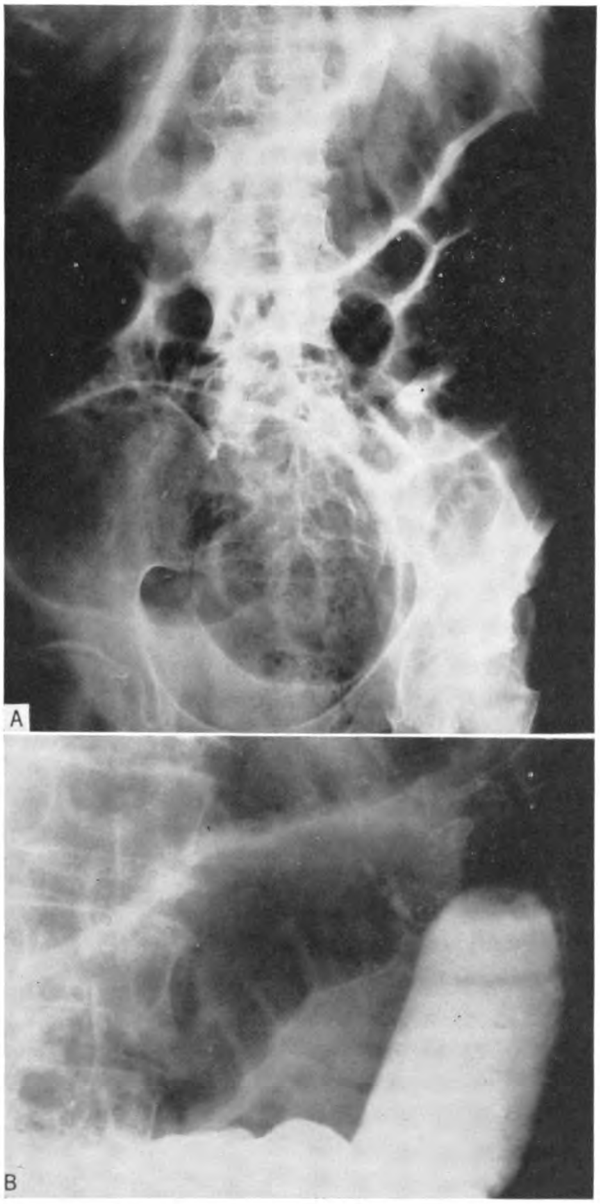

FIG. 87 —*A*, Impending cecal perforation in an 88-year-old man with a carcinoma of the splenic flexure. The cecum was paper thin at operation.

B, Same patient. Spot film showing block at splenic flexure.

two conditions. Nevertheless, considerable difficulty may arise in the differential diagnosis between diverticulitis and tumor.

Radiologically, the approach is the same as with the rest of the colon. The relative ease with which obstructive lesions can be seen is contrasted with the difficulty of finding small, asymptomatic lesions. There are a few points in identifying tumors of the sigmoid worth recounting. Occasionally, an overdistention of the rectum appears, with a relatively sudden transition to a narrowed rectosigmoid. This should arouse suspicion of an encircling lesion which can be obscured

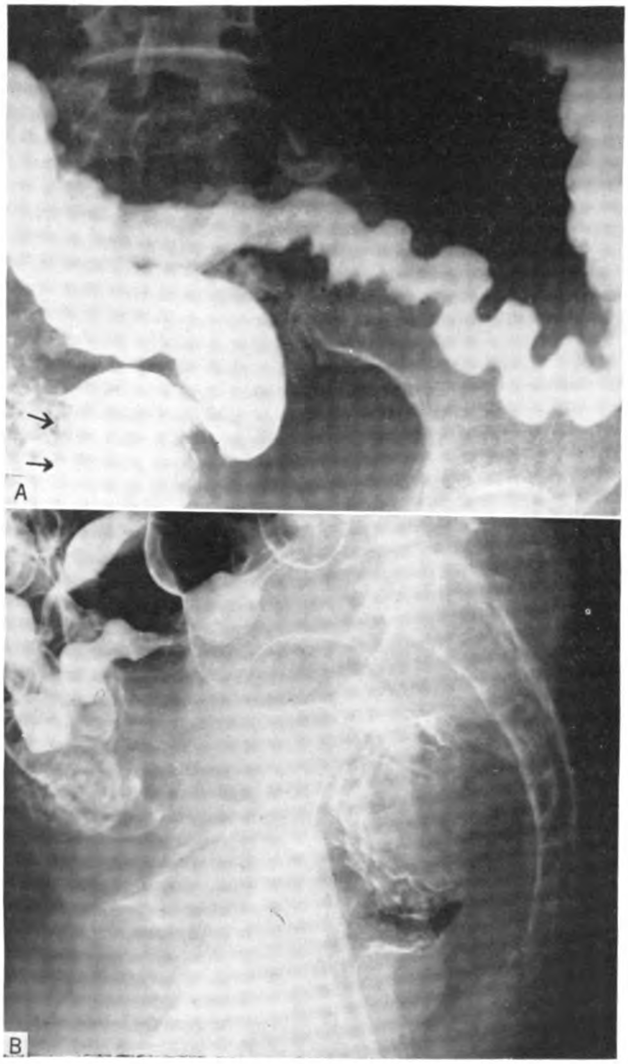

FIG. 88.—*A*, Spot roentgenogram of a 57-year-old woman with a rectal carcinoma. The left lateral wall of the rectum is rigid and deformed (arrows).

B, Same patient, lateral air contrast study. The mass is clearly identified on the posterior wall, and has extended into the retrorectal soft tissues.

by redundant bowel. A slight rigidity of the distal border of a scirrhous tumor with no obstruction occasionally points to the neoplasm. This can be seen better fluoroscopically, and spot films made as the head of the barium column meets the infiltrated area may indicate an unsuspected tumor. Marked irritability and spasm of the sigmoid sometimes simulates an infiltrating tumor. Here the variability of the picture, and study of the mucosal pattern and pliability of the wall on compression, post-evacuation and air contrast studies may be helpful. If there is any doubt, the study should be repeated. Other intra-abdominal disorders, particularly pelvic inflammations and tumors, may displace or distort the bowel, simulating intrinsic growths. This is not infrequent with ovarian carcinomas with serosal extension. Obstruction should be suspected when there is prolonged retention of a barium meal.

It is often asserted that the radiologist should not be held responsible for overlooking a neoplasm in the distal colon. The origin for this is the fact that digital and proctosigmoidoscopic examination reveal tumors here in the great majority of cases. Nevertheless, the radiologist must make every effort to identify lesions wherever they exist.

In the present series there were 6 patients with multiple tumors. Most were relatively close to one another, but one had an annular carcinoma of the sigmoid together with a large pyloric scirrhous carcinoma, and another had neoplasms both in the sigmoid and proximal transverse colon. There were 15 cases in which lesions of the distal colon were overlooked on gastrointestinal examinations, and were readily identified by barium enemas.

Infrequently intussusception of the sigmoid is secondary to polypoid lesions in the upper portion of the loop. The clinical picture in 2 such cases was left lower quadrant pain with bright red bleeding. In one the intussusception was reduced by barium enema, and in the other only a small trickle of barium passed the head of the lesion. Polypoid tumors were found in both at operation.

DIFFERENTIAL DIAGNOSIS OF COLONIC NEOPLASMS

Tumors of the cecum and ascending colon may be confused with an inflammation which contracts and distorts the bowel, or with granulomas such as ileocecal tuberculosis or actinomycosis. Deformity of the terminal ileum sometimes helps identify a nonmalignant origin of cecal and ascending colon deformities. Patients with regional enteritis, particularly older ones, sometimes have cecal deformities due to extrinsic serosal involvement or fistulous tracts. Occasionally the cecum is involved by extension from an ileitis. Diffuse ileitis extending for some distance away from the ascending colon is important in establishing this point. Occasionally, difficulty is encountered in distinguishing regional enteritis from tuberculous or actinomycotic infections of this area.

Inflammation from a perforated cecal diverticulum sometimes distorts the bowel so that a neoplasm is diagnosed. Appendiceal abscesses which are relatively encapsulated and deform the cecum likewise may simulate a carcinoma. Occasionally patients with amebiasis present an irregularity and narrowing of the cecum because of edema and spasm, and similar spastic points occur elsewhere in the bowel. As a rule amebiasis results in rather indefinite changes, and the diagnosis is best made by finding the causative organism.

Extrinsic pressure against the cecum simulating an intrinsic mass is sometimes caused by metastatic implants in the serosa from adjacent tumors such as ovarian carcinomas. Retroperitoneal tumors can elevate or displace the cecum, as may dermoids or large fibroids. These usually are sufficiently characteristic to enable a diagnosis of extrinsic displacement of bowel to be made. Occasionally encapsulated intra-abdominal effusions and abscesses produce extrinsic defects simulating tumors.

Protrusion of the ileocecal valve into the cecum may simulate an intraluminal mass. Vigorous preparatory catharsis is known to exaggerate defects produced by ileal prolapse. At times they cannot be distinguished from intracecal tumors. The filling defect varies with the site of implantation of the valve, the extent of mucosal prolapse and the size, shape and thickness of the valve lips. Abnormal ileocecal valves also may project into the cecum, but here the characteristic beaklike appearance should help make the diagnosis.

A physiological phenomenon which occasionally causes difficulty in the diagnosis of hepatic flexure tumors is spasm of Cannon's sphincter. This is not a true sphincter, but rather is a site of abnormal muscular contractions which appear because of the change in the nerve supply of the adjacent segments of hepatic flexure and transverse colon. In some patients a significant delay in passage of barium at this point occurs, at times so persistent that the examiner concludes an obstruction is present. Careful inspection of this area over a sufficiently long time, or after administering a quick-acting antispasmodic, usually discloses the transient nature of the change. Should there be any doubt, it is best to reexamine the patient.

Inflammatory lesions around the wall of the bowel such as diverticulitis or regional

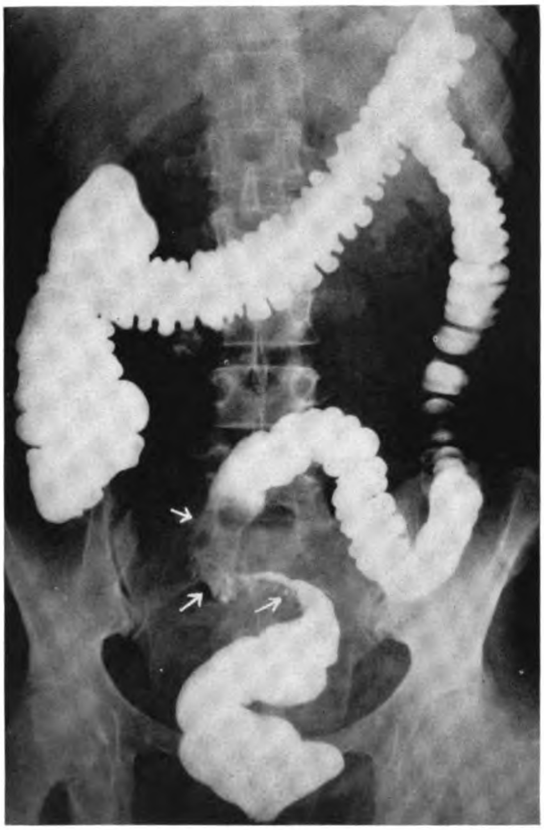

FIG. 89.—Deformity of the sigmoid in a 48-year-old woman with endometriosis (arrows). At operation the sigmoid was heavily infiltrated. The microscopic examination was reported as "looking like uterine curettings."

colitis with extension of the inflammation to the serosa also may simulate tumors. The differential diagnosis between diverticulitis and carcinoma is often difficult. Acute inflammation of the bowel may result in spasm of the circular muscles, with a resultant sawtooth pattern and tenderness on palpation. A fibrotic reaction increases the deformity, producing a narrowed lumen. The maintenance of a normal mucous membrane under such circumstances is considered against the diagnosis of malignant disease. This is often a tenuous point. The presence or absence of diverticula may help, but against this is the fact that diverticulitis may exist without visible diverticula, and cancer may be present with extensive diverticulosis. In these cases the diagnosis is best established by pathologic examination.

Endometriosis is another condition which deforms the bowel, simulating an intrinsic neoplasm. Intestinal obstruction may supervene. A history of dyspareunia, unusual bleeding, or pain on defecation at the time of menstruation sometimes is helpful. Lesions incident to endometriosis may be constricting or nonconstricting, the former simulating an annular growth. However, the mucosal folds remain intact. In the nonconstricting lesions the nodules may simulate submucosal polypoid masses. Some variation has been reported in the size of these lesions with menstruation.

Following intensive *x*-ray therapy to the pelvis, marked irritability and fibrosis of the bowel may appear which simulates tumor. In the acute phase irritability and mucosal changes are quite pronounced, with bloody mucoid diarrhea. Later changes with fibrosis predominating may be associated with irregularities suggestive of neoplasm, including intestinal obstruction. Perforation of the intestine during either the acute or fibrotic changes is followed by acute symptoms.

Differential diagnosis between ileus and obstructive lesions of the colon requires barium enema examination. It should be kept in mind that even with a nonobstructive ileus the large bowel may be so atonic that true mechanical obstruction is simulated by the hesitation in passage of barium at the splenic and hepatic flexures particularly. We saw 2 such cases recently. One was a 58-year-old man who had had a transverse colostomy done at another hospital. Subsequent *x*-ray examination showed no lesion distal to the colostomy. At necropsy 2 weeks later an unsuspected clear cell carcinoma of the kidney was found to have penetrated into the abdomen, giving rise to a diffuse carcinosis and ileus. The second patient, a 58-year-old man, had a clinical diagnosis of intestinal obstruction at the splenic flexure, and barium enema showed no passage beyond this region. However, a kidney stone was identified, and further investigation disclosed a left ureteral obstruction. The patient responded to proper urologic treatment, and the ileus cleared. A repeat barium enema examination 1 week later disclosed a normal colon.

The problem of recurrent neoplasm at an anastomotic site following resection of a tumor often is a difficult one. Here the difficulty is evaluating small deformities which may be due to the previous operation. However, the presence of a rigid focus or a polypoid defect should alert the examiner of a possible recurrence. Beal and Cornell, in reporting on 21 local recurrences, found these more often on the left side. They were of the opinion that implantations of exfoliated tumor cells and colonic papillomas both had significant roles in the development of recurrences.

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COLONIC POLYPS

Large bowel polyps are observed from infancy to old age. In children these are benign, and most often are single pedunculated growths, but also occur as multiple or sessile polyps. Usually they are without symptoms, and come to notice because of bleeding or protrusion through the anus. Anatomical studies in these children show redundancy of the mucosa. Constipation and faulty bowel habits with straining are considered etiologic agents in the production of mucosal polyps. No malignant changes in polyps have been encountered in infancy and childhood. Harris, in a review of 70 children from 1 to 12 years old, noted that most polyps appeared between 1 and 6 years of age. There is a tendency for polyps to disappear spontaneously. In many cases autoamputation occurs, which possibly explains the lower incidence after the age of 12. Andren and Frieberg commented on the spontaneous regression, suggesting that surgery often may be unnecessary.

Polyps of the colon in adults also occur as sessile or pedunculated growths. Occasionally the base of a polyp is relatively flat, and there may be a question as to whether or not a stalk is present. The head arises from the glandular epithelial layers, whereas the stalk is formed from the submucous fibrous tissue. As the head of a polyp progresses, it drags tissue behind it into the stalk. In the rectum an interesting variation occurs in the form of a villous tumor. These are flat lesions with thin, almost hair-like covering. The form of polyposis seen with pigmentation of the mucous membranes and skin, the Peutz-Jeghers syndrome, also occurs in the colon.

There appears to be a familial predisposition to the formation of polyps. These occur as single isolated lesions or in a diffuse, widely spread pattern. The correlation between the radiologic demonstration of polyps and their identification at operation and necropsy is neither constant nor favorable. Polyps are most frequently seen in the rectum and sigmoid, but appear anywhere in the colon, and when multiple, often are dissimilar in form, with sessile and pedunculated polyps intermingled.

Polyposis of the diffuse familial variety occurs as an inheritable disease transmitted to both sexes as a Mendelian dominant characteristic. Symptoms as a rule do not appear until later in life. Mayo, DeWeerd and Jackman noted that in 76.7 per cent of their 95 cases symptoms appeared between the ages of 20 and 39 years.

The most important fact about polyps, whether symptomatic or not, is their potential for malignant degeneration. As a rule, bleeding from polyps is not profuse, and patients with single lesions often have nothing more than an occasional bloody bowel movement, or the blood may appear on straining without actual stool. With diffuse polyposis loose stools or bloody diarrhea may appear. Inasmuch as many occur in the rectum or sigmoid, careful proctosigmoidoscopic examination is essential.

The radiologic investigation of the colon for polyps requires primarily a well-prepared bowel and an alert examiner. The flow of barium must be carefully observed fluoroscopically, and appropriate spot films with and without compression and of varying density should be made. The precise technic

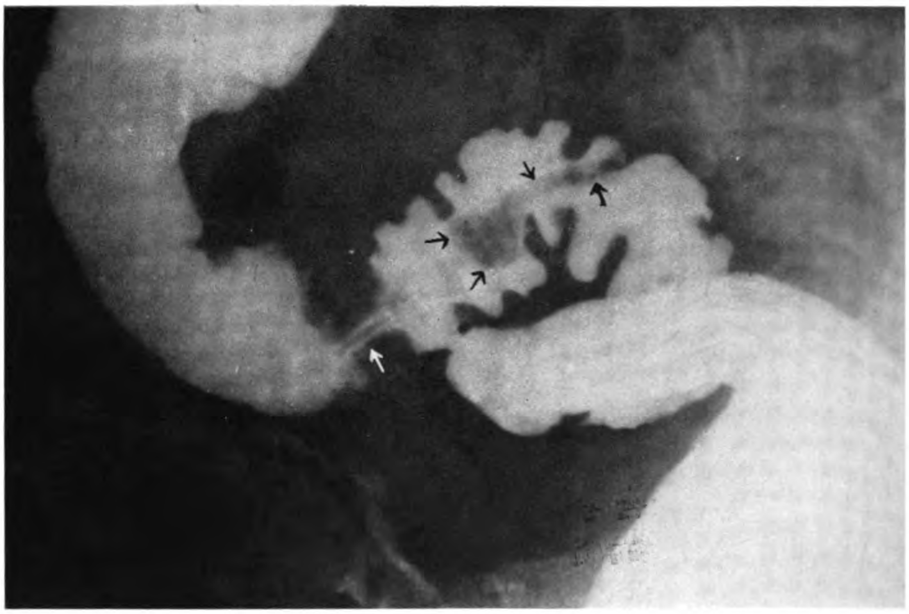

FIG. 90 —Adenomatous polyp on a stalk (arrows) in a 67-year-old man with rectal bleeding. Proximal to this is an annular carcinoma.

varies with the individual, emphasis being placed on use of a contrast medium which is not too dense, adequate spot compression studies and mucosal and air contrast examinations. No specific number of films or positions are taken. The spot roentgenograms and post-evacuation mucosal studies serve to identify pedunculated lesions by a radiolucent shadow at the head of which is a somewhat circular or ovoid area of radiolucency. The length of the stalk varies from film to film depending on its position. The longest we have seen was about 6 cm. long, but others up to 10 cm. have been reported. The presence of a stalk occasionally is helpful in deciding whether a polyp or an artefact is visible. The possibility of artefacts simulating polyps is considerable and often confusing.

Sessile polyps alter the configuration of the bowel according to their size and infiltrative tendencies. If they should be border forming, a definite irregularity is present, but if caught en-face, circular shadows of a size corresponding to the lesion appear. If a sessile polyp is relatively low, it may be obscured by the overlying barium, and in this situation high voltage technics with a rather thin mixture is advantageous. Malignant degeneration is somewhat more frequent with sessile polyps, or in the base of pedunculated polyps. It is not uncommon for a malignant lesion to be situated close to a benign one. These cannot be distinguished one from another radiologically. However, contraction of the bowel wall or irregularity along its lateral aspect increases the possibliity of malignant change. Sometimes polyps encircle the bowel, causing a partial obstruction. In familial polyposis, the tumors vary in size from a few millimeters to over 9 cm. in diameter, and histologic examination will disclose many with malignant changes while others are benign.

Infrequent lesions such as fibromas or lipomas of the bowel occur as sessile or intramural lesions simulating polyps. The pseudopolyposis associated with ulcerative colitis also causes some indecision, but the radiologic appearance of colitis is readily distinguished from true intestinal polyposis. The occurrence of localized polyposis together with segmental colitis was seen in 2 instances, both with intraluminal protrusion of the

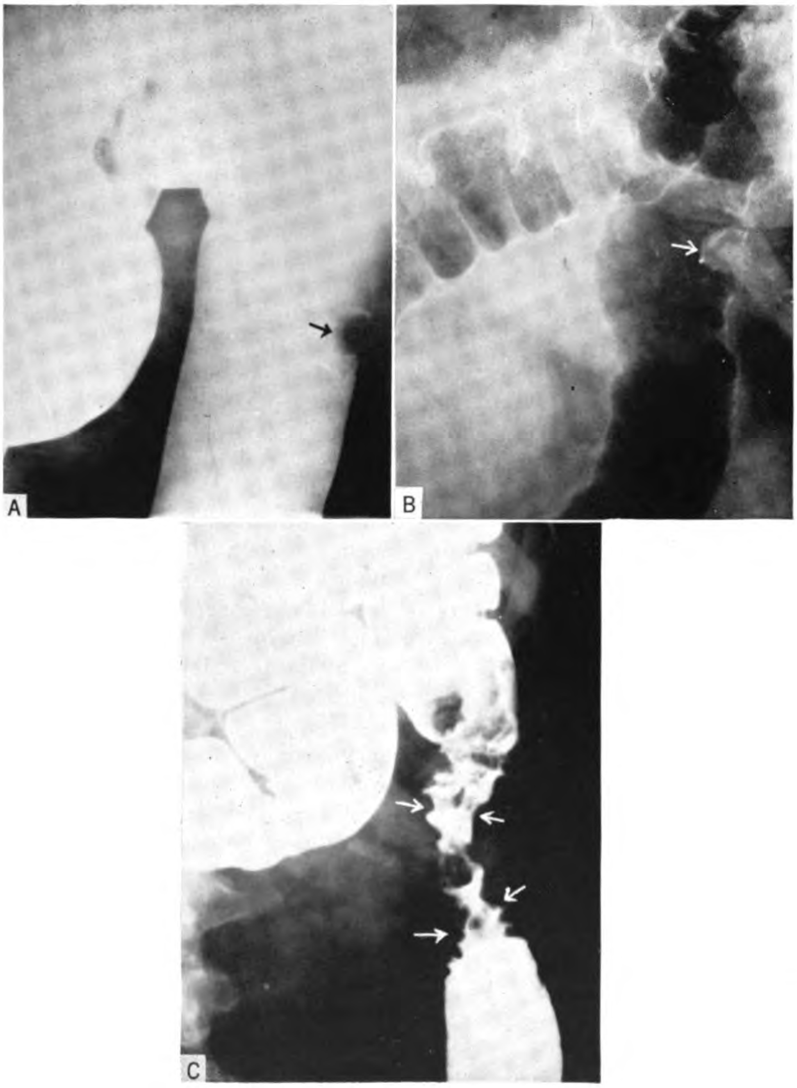

FIG. 91.—*A*, Spot film showing small sessile polyp on lateral wall of splenic flexure (arrows) in a 65-year-old woman.

B, Air contrast examination, same patient. Surgery was refused. (arrows)

C, Three years later a well-developed adenocarcinoma of the splenic flexure is present.

polyps into the bowel lumen. In both there was great difficulty in distinguishing the lesion from a tumor, and the diagnosis was made after operation. Occasionally serosal tumorous metastases or endometrial implants produce filling defects which simulate polyps.

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MESENCHYMAL TUMORS OF THE COLON

These are relatively infrequent, and cannot be distinguished from other new growths. Lymphoid tumors occur as benign polyps or malignant lymphomas. The principal site of colonic lymphoma is just above the rectal sphincter, but they occur elsewhere as single or multiple lesions, or as part of a generalized disease. Diagnosis is best established by biopsy. Helwig and Hansen noted that a lymphoid tumor of the colon other than in the rectum or anus was more likely to be malignant. A favorable response to *x*-ray therapy in one of our patients lasted for 2 years. After that time a generalized involvement developed in the chest, peripheral lymph nodes, stomach and small intestine.

Leiomyomas and leiomyosarcomas also appear in the colon, particularly the rectum. Osmond and Mautz collected 20 cases from the literature, and added another in a 73-year-old woman in whom a spherical mass produced narrowing and displacement of the upper rectum. These tumors are rapidly growing, and originate in the subserous, muscular or submucosal layers, and grow either into the lumen of the bowel, externally into the pericolonic structures or project to either side of the bowel wall. They tend to be encapsulated, and ulceration of the overlying mucosa occurs with bleeding. Recurrence after removal is frequent, with extension into the pelvis and adjacent lymph nodes. These tumors produce abnormal bowel patterns, discomfort and bleeding. There may be difficulty in differentiating them from retrorectal tumors such as chordomas, teratomas, neurofibromas or dermoids. Occasionally bone tumors such as osteogenic sarcomas, fibrosarcomas or giant cell tumors originate in the retrorectal area and displace the colon.

Another unusual colonic tumor is the lymphangioma. Arnett and Friedman reported the tenth such case, which appeared as a fluctuant intramural mass without mucosal change on a barium enema examination. They noted that of the 9 lymphangiomas in the gastrointestinal tract mentioned in the English literature, 6 arose in the jejunum or ileum and 3 in the rectum. No specific roentgenologic characteristics are described. Puppell and Morris assumed lymphatic obstruction causing increased pressure within the lymph nodes to be the causative mechanism.

Lipomas of the colon are encountered more frequently. Stapleton found 130 cases in his review. These tumors usually are situated in the cecum and ascending colon, but occur elsewhere as well. The symptoms and signs incident to them depend on their size, the possibility of torsion, obstruction or intussusception, the latter occurring fairly often. Ryrberg suggested that the relatively radiolucent character of lipomas might some-

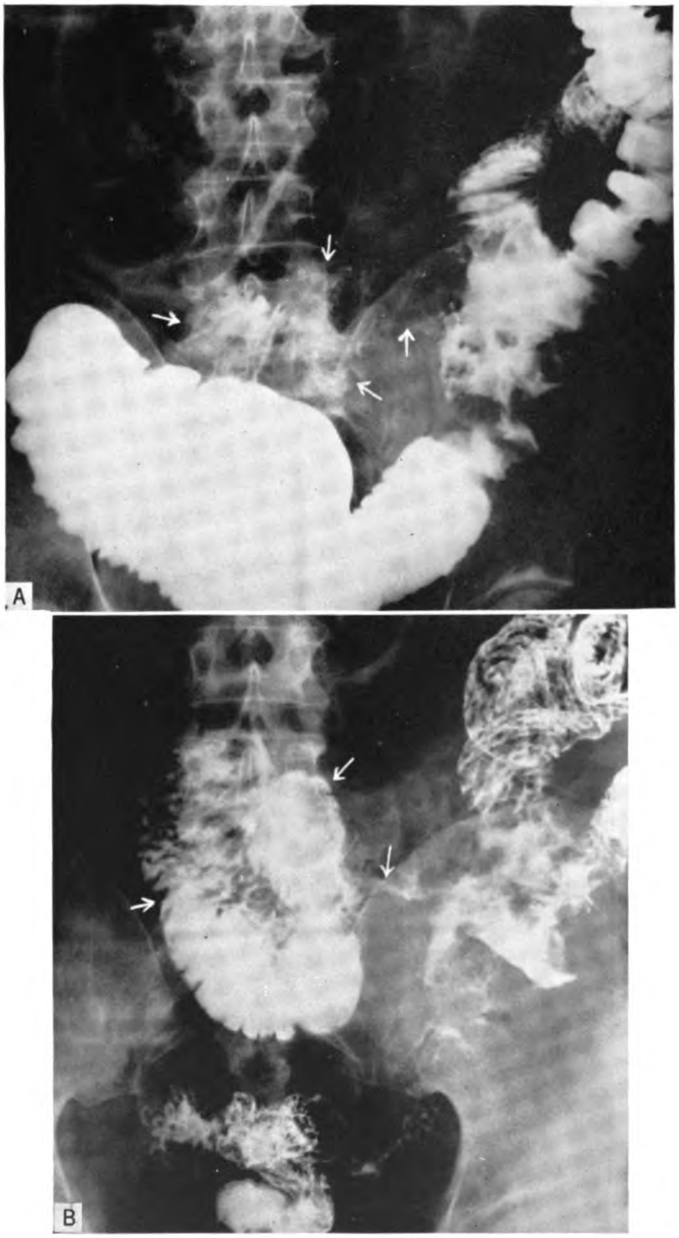

FIG. 92.—*A*, Lymphosarcoma of the sigmoid colon in a 42-year-old man with lower abdominal pain and rectal bleeding. A large filling defect is present, and barium enters the mid-ileum through an ileocolic fistula (arrows).

B, Same patient, post-evacuation film. The colon has emptied, but the lesser bowel contains more barium than on the preceding study (arrows).

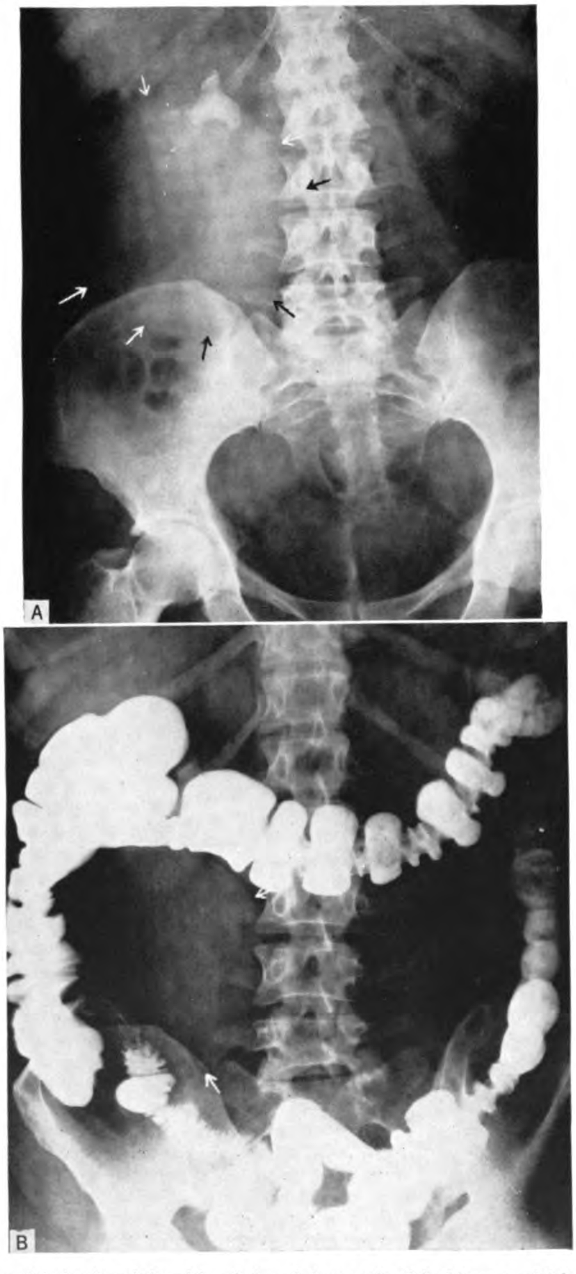

FIG. 93.—*A*, A mass was felt in the right side of the abdomen in this 53-year-old female who had symptoms of a right renal calculus. Scout film shows a large kidney stone and a well-outlined ovoid mass (arrows). *B*, Barium enema shows an extrinsic defect in the medial wall of the descending colon. At operation a fibroma originating from the bowel wall was removed. The mucosa was intact.

times be helpful in diagnosis, and also noted that these masses are associated with symptoms suggestive of malignancy such as weight loss, melena, anemia and lassitude. The diagnosis is best established at operation.

Fibromas also occur within the wall of the bowel, and occasionally become fibrosarcomas. These tumors extend extrinsically, giving rise to large palpable masses. One such case was observed by us in a 53-year-old man who came in for renal roentgenographic examination because of a right ureteral calculus. A mass was seen in the right side of the abdomen, and on barium enema this was observed to displace the ascending colon laterally. At operation a fibroma originating in the medial wall of the ascending colon was removed.

Melanomas occur occasionally, especially in the anorectal area. These usually are small and tend to be covered by normal mucosa. The diagnosis is best established at proctosigmoidoscopy. Only occasionally can they be identified radiologically as polypoid lesions.

Carcinoids are sometimes found in the colon, particularly in the anorectal region. Mayo and McKee noted that 66 cases had been reported, and added 2 more. These tumors tended to invade the mucosa and muscularis, were accompanied by bloody stools, tenesmus and pain, and were malignant. Most were less than 1 cm. in diameter, and were usually diagnosed proctoscopically. Freund surveyed 303 cases, and he too noted that a rectal carcinoid could not be distinguished grossly from any other benign or malignant submucosal nodule or adenoma. Carcinoids also have been identified elsewhere in the colon, including the cecum and sigmoid.

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CARCINOMA OF THE BILIARY TRACT

Adenocarcinoma is the most frequent neoplasm of the gallbladder. This occurs as a scirrhous or medullary lesion, the former as a hard, thick contracted biliary sac which often simulates chronic cholecystitis. The latter is a more proliferative tumor, occasionally papillary in type, and rarely mucoid. Occasionally a combination of squamous and adenocarcinoma referred to as adenocanthoma is seen. In about 10 per cent of cases, a squamous cell carcinoma of scirrhous form appears, believed due to metaplasia of chronically inflamed gallbladder mucosa.

Gallbladder carcinomas are responsible for about 4 per cent of deaths from carcinoma. Another indication of its incidence is its association with from 1 to 8 per cent of all gallbladder operations. The disease occurs 4 times more often in women, usually in middle or late life. Previous inflammation of long duration, with or without gallstones, is considered a precursor. A normal gallbladder with a carcinoma is rare. The question as to whether gallstones do or do not predispose to malignant change is still under discussion.

Cancer of the gallbladder arises insidiously, with early symptoms of vague abdominal

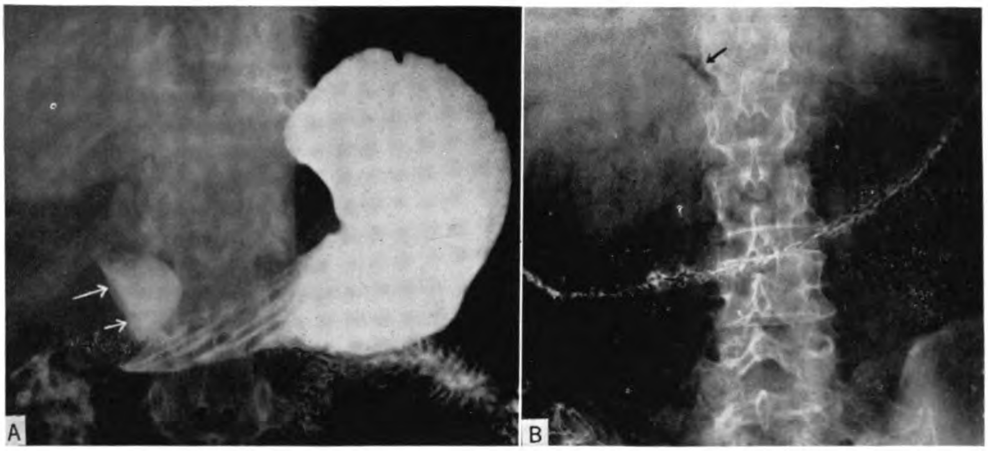

FIG. 94.—*A*, Perforating carcinoma of the gallbladder in a 60-year-old woman deflecting lateral aspect of duodenum (arrows).

B, Same patient, post-evacuation barium enema film. The colon shows a slight depression of the proximal transverse colon. The duodenal bulb, which contains some air, is deflected (arrows). Lateral to this are numerous gas bubbles, found to be due to necrosis within the tumor with colon bacillus infection.

disorders, often insufficient to cause concern. Later the symptoms are referrable to obstruction of the biliary passages and invasion of nearby viscera. Fistula formation to adjacent organs may occur, or there may be perforation with biliary peritonitis. Bleeding in a degenerated tumor also may cause relatively acute symptoms. Jaundice appears with blockage of the common bile duct or sufficient intrahepatic invasion. Metastases are by way of the lymphatics, the systemic circulation and by direct extension.

The radiologic diagnosis of carcinoma of the gallbladder is difficult. Oral cholecystography usually shows only no or poor visualization of the sac, and intravenous cholangiography offers no specific assistance. With perforation and concomitant inflammation a mass may be palpated, and vaguely demonstrated on plain film examination. If the lesion involves the cystic duct, hydropic dilatation of the gallbladder may be identified as a well-defined saccular opacity. Extension of the tumor into the porta hepatis is followed in some by duodenal obstruction, with dilatation and gastric retention. Displacement of the duodenal loop and extrinsic pressure defects on the stomach may appear. With a massive lesion there is often displacement and invasion of the pylorus or duodenum. It is not at all uncommon to find the upper gastrointestinal tract normal. In cases in which the gallbladder fills during intravenous cholangiography a filling defect of a polypoid nature is sometimes seen.

Papillomas of epithelial origin in the gallbladder have been reported approximately once in every 100 cholecystectomies. These may be multiple, and sometimes are confused with nonopaque stones. Their location and size are not diagnostic, and there is no particular site of predilection for their occurrence. According to Kane, Brown and Hoerr, no symptoms are attributable to this condition. In their description of 8 verified cases gallstones also were found in 3, cholesterosis in 1 and 2 had chronic cholecystitis. Tabah and McNeer recently reported 4 cases, in 3 of which there were foci of noninfiltrating carcinoma *in situ*. These are believed to be the first such cases recorded, and the authors suggested cholecystectomy on this basis. Oakland pointed out that the term papilloma had been applied to polypoid conditions of the gallbladder histologically similar to the cholesterol-laden villi of the strawberry gallbladder as well as to true new growths. Similar lesions also occur as the end result of chronic inflammation. In a recent review of tumors and pseudotumors of the gallbladder, Jones and Walker reported 28 cases in 1000 patients, of which 16 were cholesterol polyps, 4 inflammatory polyps, 3 cholecystitis glandularis proliferans and 5 congenital malformations.

Radiologically these lesions appear as fixed, small, relatively circumscribed filling defects in visualized gallbladders. They can be distinguished from stones by their relative immobility, but one cannot pass on the histologic nature of the lesion without microscopic study.

Fibromas of the gallbladder are uncommon. We saw one such lesion situated subserosally in a 55-year-old woman whose cholecystograms revealed a semicircular filling defect in the periphery of the gallbladder. No symptoms were attributed to this, but the lesion was removed on the suspicion that it might be malignant.

Neoplasms of the bile ducts have been observed in the cystic duct, the common hepatic duct and the common bile duct. Of the 63 cases reported by Kuwayti, 8 occurred in the cystic duct, 15 in the common hepatic duct, 11 at the junction of the cystic and common bile ducts, and 23 in the common bile duct. Six involved the left hepatic duct, while there were no cases in which the right hepatic duct was affected. The tumors which originate in the lesser biliary radicles cannot be accurately distinguished from primary liver cancer, and are not considered here.

Tumors of the bile ducts occur more often in males at or beyond middle life, although occasional cases are reported in relatively young people. The clinical picture is predicated on biliary obstruction and consequent jaundice. This tumor has a tendency to spread locally. As a rule the course is relatively rapid, with terminal hepatic failure and coma. The incidence of gallstones in association with cancer of the bile ducts is about 40 to 50 per cent, but there is considerable doubt as to a relationship between these conditions. Mild ascites occurs in about 25 per cent, usually insufficient to be noted clinically or radiologically.

The radiologic diagnosis cannot be made with confidence. The presence of a hydropic gallbladder in a jaundiced patient sometimes brings this diagnosis to mind. Operative cholangiography is necessary, particularly if the lesion involves an intrahepatic duct.

CARCINOMA OF THE AMPULLA OF VATER

When ampullary carcinomas are small, they may be absolutely asymptomatic. However, with growth of the tumor there is progressive weakness, weight loss, epigastric distress and sometimes back pain radiating to the right is noted. As a rule, however, the patient first comes for care when jaundice develops. This may be intermittent, and is accompanied frequently by chills and fever. With recurrent biliary obstruction and infection, the chills, fever, sweats and abdominal pain may be quite severe. In the intervals of relief from jaundice, these symptoms abate. Von Eigen reported a case of intermittent fever for 2 years due to an ampullary carcinoma with recurrent biliary obstruction and secondary infection. This syndrome is not specific, and occurs with other lesions such as tumors of the pancreas or bile ducts, strictures of the bile ducts, cystic duct obstructions and sometimes cholecystitis.

The radiographic diagnosis of ampullary carcinoma can be more accurate than other biliary tract neoplasms. If there is intermittent jaundice, oral cholecystography discloses in some patients visualization of a dilated gallbladder. Intravenous cholangiography also is occasionally useful in identifying dilatation of the common duct and of the gallbladder. The association of such dilation with filling defects at the distal end of the common duct may not be distinguishable from an impacted stone about which drainage is possible. Nevertheless, the decision for an early operation is facilitated.

Investigation of the upper gastrointestinal tract is helpful. The tumor sometimes is identified directly as a small filling defect in the descending duodenum. Should there be dilatation of the common duct, its impression against the inner aspect of the descending duodenum produces a relatively shallow, somewhat scalloped impression. With larger lesions spread of tumor around the duodenal mucosa may encircle the duodenum, with dilatation above the obstructive site. Some-

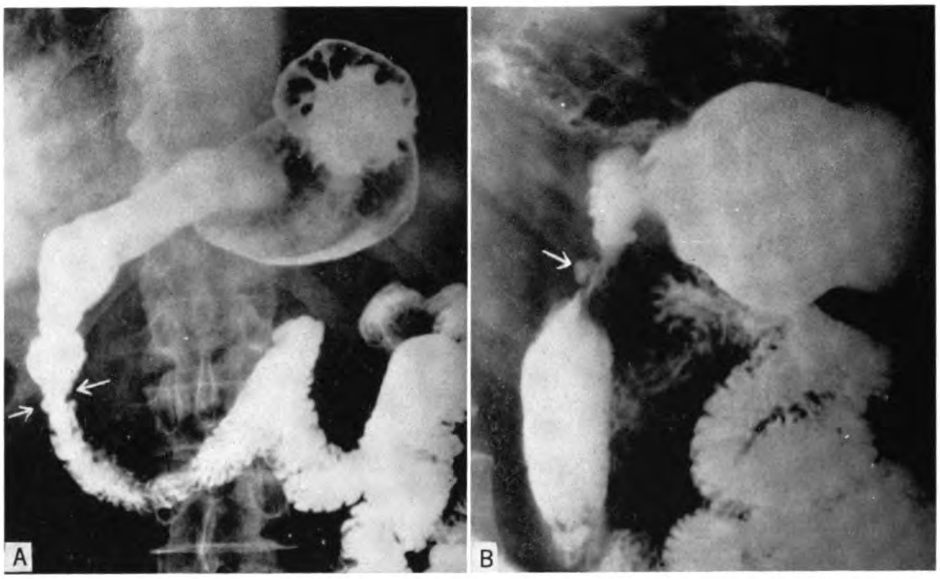

FIG. 95.—*A*, Carcinoma of the ampulla of Vater in a 55-year-old man with jaundice. Supine film shows a narrowing at the midportion of the descending duodenum (arrows).

B, Same patient, right lateral prone view shows the deformity of the descending duodenum to better advantage (arrows). At operation the tumor encircled the duodenum.

times tumor extends down the duodenum and cannot be distinguished from duodenal carcinoma. These tumors also extend extrinsically into the porta hepatis, and then are indistinguishable from pancreatic newgrowths. Occasionally the diagnosis is made by operative cholangiography or postoperative retrograde cholangiography, when small filling defects at the distal end of a dilated common duct may lead to the correct diagnosis.

Occasionally a carcinoma of the ampulla of Vater is not identified until after cholecystectomy for gallstones. In five of our cases postoperative retrograde cholangiograms were made because of persistent jaundice and biliary drainage. The filling defects at the distal end of the common duct were verified at subsequent operation.

The importance of early diagnosis is highlighted by the possibility of long term palliation after adequate surgery. Recently Swan and Pfister reported a case of 9-year-survival in an elderly patient, and cited others with similar results.

Benign lesions at the ampulla also have been described. Smith and Blakemore re-

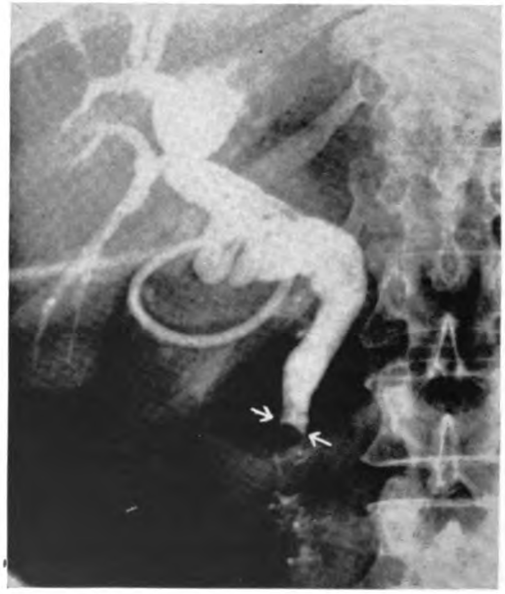

FIG. 96.—Retrograde cholangiogram showing defect in distal end of common duct due to ampullary carcinoma (arrows) in a 74-year-old woman. Cholecystectomy had been done 20 years before. Fever, chills, epigastric pain radiating to the back was followed by jaundice. Diagnosis confirmed at operation.

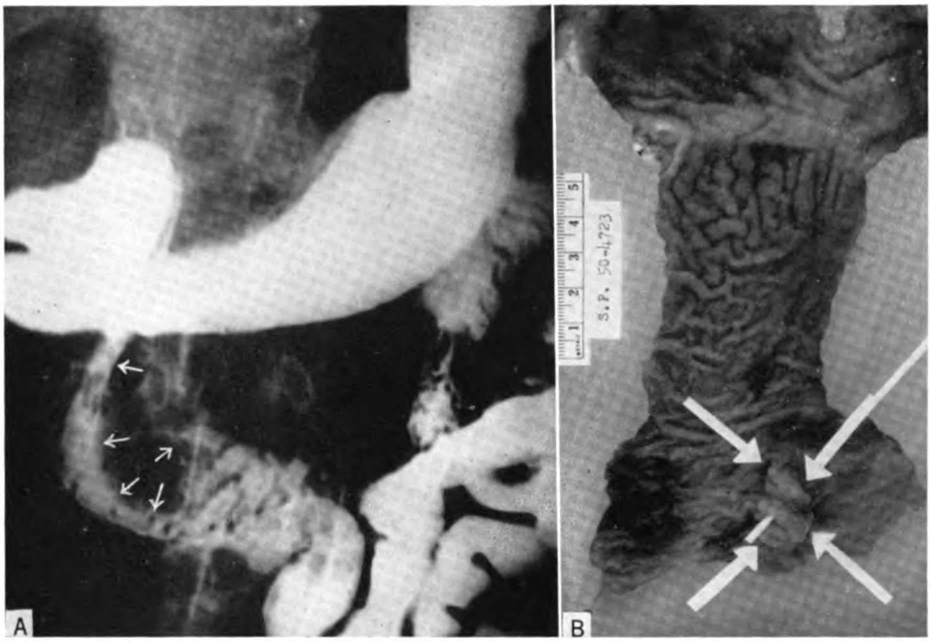

FIG. 97.—*A*, Ampullary carcinoma in a 52-year-old man with painless jaundice for 3 weeks. A circular defect is present in the mid-duodenum (arrows), and an impression is present in the inner aspect of the duodenum above this.

B, Specimen showing ampullary carcinoma. The impression into the duodenum was due to a dilated common bile duct.

ported a case in which a postoperative cholangiogram revealed a filling defect at the duodenal end of the common duct which was interpreted as a stone. This persisted for 11 days, and operation disclosed a polyp 1 cm. in diameter at the ampulla.

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CANCER OF THE LIVER

The most common malignant liver tumor is the hepatoma, or liver cell cancer. Malignant disease also arises from the smaller bile ducts, designated as cholangio-carcinoma, and rarely a sarcoma is seen. While these are rare in the United States, they are frequent elsewhere, particularly in the Orient. It is believed that preexisting inflammation predisposes toward their development, and association with cirrhosis is relatively frequent. Cohn and St. Raymond reported cirrhosis in about half the patients who had come to necropsy, but considered the overall incidence about 25 per cent. While liver cell carcinomas occur more frequently in middle-aged males, they also are encountered in infancy. Edmondson designated them as the commonest carcinoma of childhood, with males up to 2 years old affected most often. He commented on the fact that in regions where liver carcinoma occurs frequently there is an absence of reported cases of this tumor in childhood.

The clinical and radiographic picture of primary carcinoma of the liver in infancy and childhood is dominated by a firm lobulated mass in the right upper abdomen. Occasionally these tumors contain osteoid, and spotty calcifications may be visible on scout films. Embryonal cell carcinomas of the liver are biologically comparable to Wilm's tumors, and the prognosis for both is poor. Neuroblastomas are distinguished from primary liver tumors by their tendency to displace the right kidney downward and laterally, with calcifications in the adrenal area. Renal tumors displace the colon anteriorly and downward, while large liver tumors displace the bowel downward and to the left. It should be kept in mind that diffuse enlargement of the liver often is produced by other diseases such as von Gierke's disease, leukemia, lymphoma and rarely amyloid disease, and that visceral displacement occurs because of abdominal and retroperitoneal tumors also.

The clinical identification of primary cancer of the liver in adults is difficult. Cohn and St. Raymond indicated the scope of this problem by listing in their Table 8 the extensive range of clinical conditions which simulate this disease. As a rule, the diagnosis is one of exclusion, and probably is established best surgically. Jaundice is relatively common, occurring in about half the patients. Ascites also is fairly frequent. Concomitant cirrhosis of the liver may mask a malignant hepatoma, with gastroesophageal varices and ascites the outstanding features. The association of primary carcinoma of the liver with hemochromatosis was mentioned by Warren and Drake.

The radiologic diagnosis of liver cancer depends on the extent of the hepatomegaly and its effects on the adjacent viscera. The diaphragm may be elevated and its motion restricted. Infrequently a reactive pleural process appears in the right lower lung field. When damage to the liver parenchyma is not excessive, oral cholecystography sometimes reveals a normally functioning gallbladder. If the liver is enlarged displacement of the biliary sac is produced. Upper gastrointestinal tract examination discloses displacement of the stomach and duodenum, as well as depression of the transverse colon and hepatic flexure, reflecting the hepatomegaly.

Infrequently a liver tumor extends through the diaphragm and involves the thoracic cavity. Adams reported 2 cases in which the symptoms were primarily of thoracic origin. Elevation of the left diaphragm and hydrothorax was interpreted as a malignancy of the left lung in one. At bronchoscopy, atelectasis of the left lower lobe from extrinsic pressure on the bronchus was noted, with no visible tumor. Exploratory thoracotomy disclosed a dense hard mass involving the diaphragm, the left lower lobe and lingula and the left lobe of the liver. This was found to be due to a hepatoma originating in the left lobe of the liver.

The prognosis for liver tumors is poor even if extensive surgery is performed. Packard and Palmer noted that no cases survived over 22 months even though removal was considered adequate.

Benign tumors of the liver occurring during infancy are referred to as hamartomas, isolated solitary nodular hyperplasia of the liver, focal cirrhosis and benign adenomas. Multiple nodular hyperplasia gives rise to large masses attributed to proliferative changes which follow acute necrosis of the

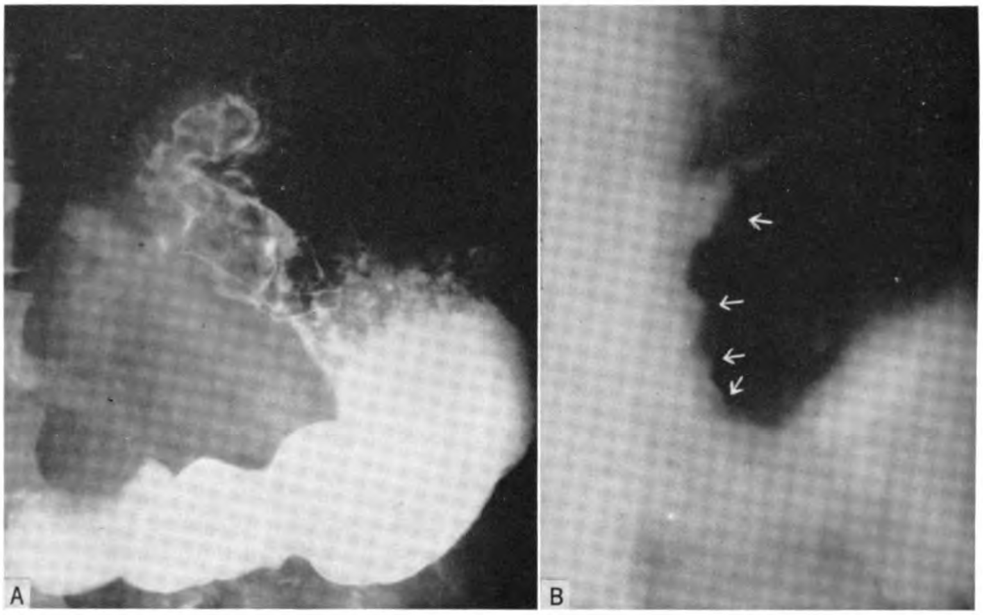

FIG. 98.—*A*, Filling defect in the stomach gas bubble in a 55-year-old man with cirrhosis and a hepatoma *B*, Same patient, air contrast study with body section technic. The nodular defects were due to large esophagogastric varices.

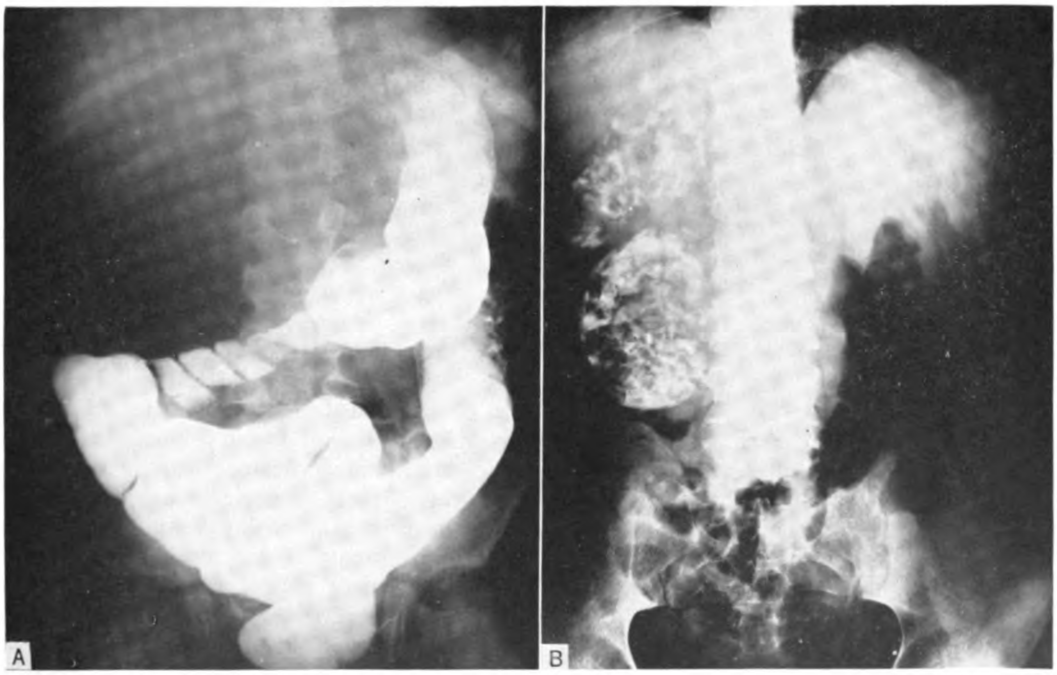

FIG. 99.—*A*, Intrahepatic tumor in a 9-year-old boy. Note the impression against the transverse colon. *B*, Same patient, 15 years later, after *x*-ray therapy given after operation identified the mass as a hemangioblastoma.

liver. Congenital cysts of the liver are seen in early infancy. The subject was reviewed extensively by Edmondson.

A relatively common benign lesion is hemangioma of the liver. This occurs as a single large mass or as diffuse multiple cavernous hemangiomas. In infancy, hemangioendotheliomas are relatively more frequent than cavernous hemangiomas. These sometimes become progressively larger and rupture, causing intraperitoneal hemorrhage. Extrahepatic hemorrhagic nodules occur in about a third of these cases. These are considered independent growths rather than metastases. Large solitary hemangioendotheliomas may be resectable, and *x*-ray therapy also can be effective. One of our cases was followed for over 15 years after *x*-ray therapy, the end result being extensive calcification of the tumor. Calcification may be due to lime salt deposit in areas of necrosis within the tumor.

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CANCER OF THE PANCREAS

Pancreatic neoplasms are predominantly of the duct cell carcinoma variety, although squamous cell, colloid and papillary cystadenocarcinomas also occur. This disease affects males and females equally, and is more common in the middle years. In about 70 per cent of cases the head of the pancreas is affected, and in the remainder the tail and body are primarily involved. A clearly-defined demarcation is infrequent, and tumor extends from one area into another quite often.

The clinical picture of pancreatic carcinoma is variable, particularly in its early stages before compressive effects occur. The patients complain of intermittent mid-abdominal pain radiating through the back. The usual symptoms of a chronic debilitating disease is slow in onset, and months may elapse before definite symptoms appear. A sudden onset of diabetes sometimes presages pancreatic carcinoma. Weight loss and emaciation appear, and vague gastrointestinal disturbances and occasionally diarrhea have all been observed. Thrombophlebitis may occur as a precursor, more often with body and tail tumors. As the tumor enlarges, it impresses the inner aspect of the duodenum and the common bile duct, and also extends toward the stomach, the transverse colon, and the retroperitoneal lymph nodes. Gastrointestinal symptoms increase in severity, simulating duodenal ulcer or gallbladder disease. Indeed, the neoplasm may ulcerate through the stomach or duodenum and produce hematemesis as well as the pain of peptic ulcer. With extension to lymph nodes near the cardio-esophageal junction dysphagia sometimes is noted.

A most interesting form of pancreatic neoplasm has been brought into prominence by the work of Zollinger and Ellison. This is the so-called ulcerogenic tumor of the pancreas, associated with fulminating peptic ulceration of the stomach, duodenum and jejunum. Marked gastric hypersecretion and hyperacidity are accompanied by recurrent ulceration after treatment. The clinical entity consists of hypersecretion, hyperacidity and atypical peptic ulceration associated with non-insulin producing islet cell tumors. In a review by Ellison, 24 instances of ulcerogenic tumors of the pancreatic islets were summarized. Fourteen were single and 10 were multiple. Tumors were found

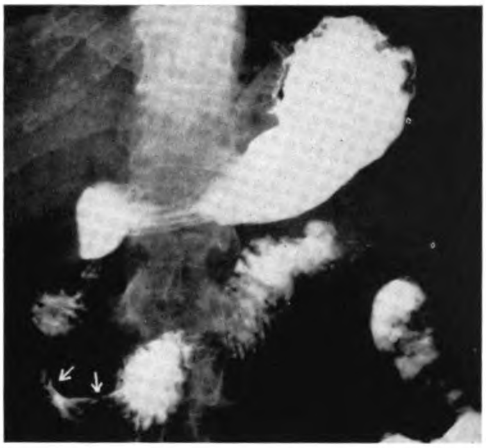

FIG. 100—Carcinoma of the head of the pancreas in a 61-year-old man with pruritus, anorexia and weight loss. Note the defect in the second and third portions of the duodenum with straightening of the walls and flattening of the mucosal folds (arrows).

scattered throughout the pancreas in 3, in the head and body in 2, and the body and/or the tail in 12. The head of the pancreas alone was involved in 4. One tumor arose in aberrant pancreatic tissue in the gastric wall. Ninteen were malignant. The rate of growth of the tumor was slow, and peptic ulcerations were the predominant manifestations in all patients. Eleven of the 19 tumors had spread beyond the pancreas. These tumors usually were small, and their identity established at operation. There is no known radiologic approach to their direct identification.

The radiologic diagnosis of pancreatic neoplasms is best understood when their size, location and extension are kept in mind and correlated with the patients' symptoms. In a review of 50 of our verified cases, no constant picture was found. However, some defect was observed in the duodenal bulb or loop, or the pylorus in more than 80 per cent. The effect of compression and obstruction of the common bile duct by tumor was reflected in the size of the gallbladder and the common duct. Widening of the duodenal loop was encountered in only 10 per cent of our cases. Other signs included impressions into the upper aspect of the duodenal loop above the ampulla of Vater, similar impressions against the inferior portion of the duodenal loop, and constriction and narrowing of the mid-duodenum, associated with extension of tumor around this area. In six cases the duodenal loop was small and compressed. At operation this was found to be due to elevation of the loop by a pancreatic mass. Impression into the inferior aspect of the stomach was seen rather frequently, particularly with large lesions. In some the duodenal loop was deformed, so that the impression into the inferior aspect of the antrum was accompanied by a smooth identation in the inferior aspect of the duodenal loop.

Direct extension of tumor into the serosa of the stomach and duodenum was not infrequent. In 3 of our cases the initial *x*-ray diagnosis was pyloric carcinoma. In 2 patients jaundice was present, and hepatic metastases or biliary obstruction was postulated. Actual pyloroduodenal obstruction with gastric retention occurred in 3 patients. In one an obstruction in the mid-duodenum was associated with pronounced gastric dilatation. In another the duodenal obstruction was such that at operation and subsequently at necropsy only a small channel remained, yet this patient's stomach emptied fairly well. The degree of deformity was less than might have been anticipated from the size of the tumor. In 1 case a duodenocolic fistula formed through a pancreatic tumor which encircled the duodenum and entered the transverse colon.

In 6 cases the duodenal bulb was so irregular and deformed that the diagnosis o duodenal ulcer was considered. In 2 the duodenal deformity was associated with nodular implants which suggested the presence of extrinsic nodules. Inasmuch as the duodenal loop was deformed, nodular extension of a pancreatic tumor was considered likely, and this was confirmed at operation.

As emphasized by Hodes, Pendergrass and Winston, the mucosal pattern and motor function of the duodenum is altered. In our cases the mucosal folds often were thickened and irregular. Retroperistaltic activity in the duodenum was frequently observed fluoroscopically.

The inverted "3" sign described by Frostberg was relatively infrequent in this series. This sign is attributed by Stiennon to enlargement of the papilla of Vater, with the upper limb of the "3" produced by enlarge-

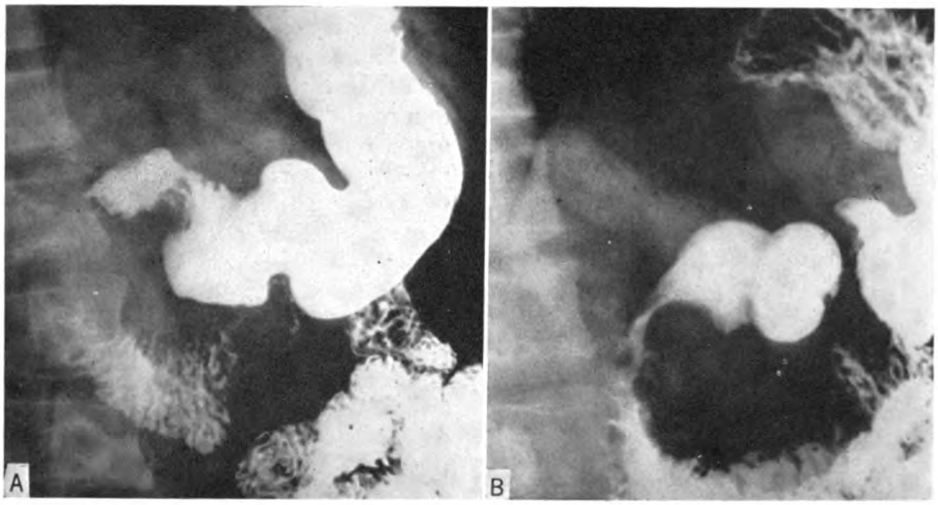

FIG. 101.—*A*, Carcinoma of the head of the pancreas. Right oblique film shows no filling of upper portion of descending duodenum.

B, Same patient about 5 minutes later. The duodenum is filled and a narrowing and flattening of its upper half is present.

ment of the minor papilla while the lower, and usually the larger limb, represents enlargement of the major papilla. The central bar results from pocketing of barium between the two enlarged papillæ and the medial duodenal wall. He believed the most common cause for this is papillary edema. The difficulty in differentiating large malignancies of the papilla of Vater from carcinoma of the head of the pancreas is considerable, both clinically and radiologically, particularly with small pancreatic tumors.

Occasionally the correct diagnosis is facilitated by identification of a dilated common duct and gallbladder caused by compression of the distal end of the common duct. Not infrequently, even without jaundice, oral cholecystography shows no visualization of the gallbladder. Impression of a dilated gallbladder against the superior aspect of the duodenal bulb is a rather variable change, and is affected by alterations in the relative positions of the viscera in the various positions. In 10 of our cases such duodenal compression was noted best with the patient in the supine oblique positions. A dilated common bile duct also may impinge against the inner aspect of the duodenal loop at its midportion, giving rise to a shallow, sometimes scalloped defect.

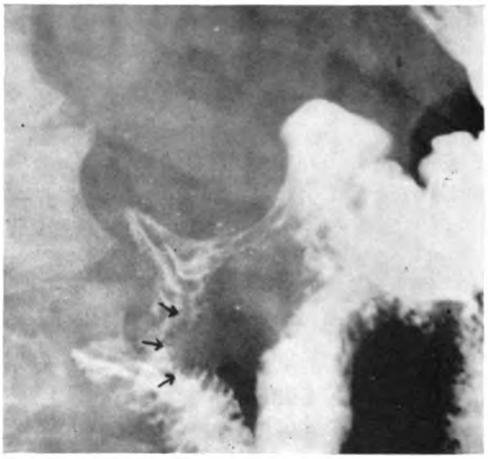

FIG. 102.—Carcinoma of the head of the pancreas in a 65-year-old man with painless jaundice, mass in right upper quadrant and pain. The indentation into the duodenum is due to a dilated gallbladder. Beneath this (arrows) is an indentation into the duodenal loop due to impression by the dilated common duct, which was about the same size as the jejunum.

An interesting change sometimes seen with carcinoma of the pancreas is a relative delay in the passage of barium through the lesser bowel, with retention there over a 5-hour period. On necropsy in 5 patients in whom this occurred, no obstructive lesions were

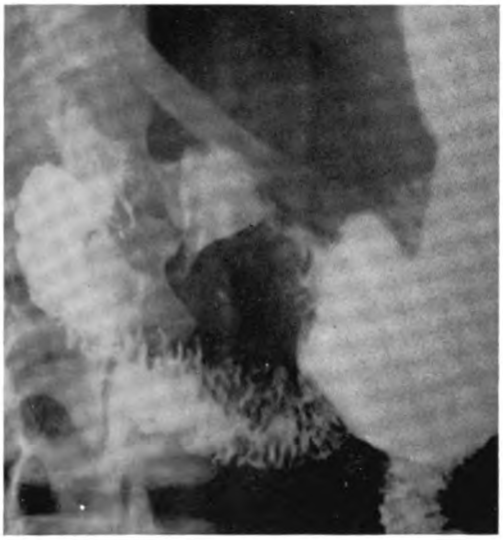

FIG. 103.—Carcinoma of head of pancreas infiltrating into pyloric antrum. Patient was a 49-year-old man with abdominal pain, anorexia, weight loss, fatty stools and diarrhea. At operation the pylorus was infiltrated and fixed, and tumor extended into the root of mesentery.

present. The cause for this delay may rest with impaired duodenal muscular function as well as possibly diminished caliber of the duodenum.

In an evaluation of the diagnostic criteria for carcinoma of the pancreas, estimates of from 40 to 50 per cent of positive findings are available. Dashiell and Palmer noted that suggestive radiological evidence occurred in 48.6 per cent of 72 patients examined. Broadbent and Kerman stated that a correct diagnosis could be made in 53.9 per cent. Even on review after knowing the diagnosis, 21.1 per cent were regarded as negative, and in 25 per cent abnormal changes were noted but a definite radiologic diagnosis was not possible.

Carcinoma of the body and tail of the pancreas infrequently is accompanied by jaundice, but more often presents severe episodic pain, rapid and extreme emaciation, and in about half the cases, a palpable abdominal mass. This tumor may develop rapidly. In one verified instance the patient complained of weight loss and mid-abdominal pain radiating to the back, but nothing pathological was found radiologically or on physical examination. Yet 4 months later he returned with a mass which filled the upper abdomen and which was easily demonstrated radiographically. Once in a while these tumors are mistaken for carcinomas of the colon because of their impression into the transverse colon. Extension to the pylorus sometimes cannot be told apart from antral carcinomas. Tumors of the body and tail of the pancreas are known to mimic those of the cardia and pars media. If the lesion is sufficiently large it can cause dysphagia. In a case reported by Marks, Weingarten and Gerst, a carcinoma of the tail of the pancreas was associated with bleeding gastric varices secondary to pressure on the venous channels of the spleen. Chamberlin and Imber noted changes on intravenous urograms consisting of varying compression deformities of the left renal pelvis, displacement of the proximal portion of the ureter laterally or posteriorly, and occasionally posterior kidney displacement as seen on lateral pyelograms. These tumors also displace the splenic flexure, and sometimes impinge on it sufficiently to imitate a tumor.

PANCREATIC CYSTS

Pancreatic cysts usually occur as benign cystadenomas, although cystadenocarcinomas also are seen. Dermoid or teratomatous cysts are infrequent. Congenital cysts are more common, and often are associated with polycystic disease elsewhere. These are neither neoplastic nor of the retention variety. Parasitic cysts are fairly frequent. Most frequent are retention cysts resulting from partial obstruction of the major pancreatic ducts, usually following chronic pancreatitis. Fibrocystic disease of the pancreas usually is not characterized by gross cystic formations.

Cystadenomas of the pancreas occur more in females. The theories of their origin include displacement of embryonic cells with neoplastic proliferation of pancreatic epithelium. The tumor is covered by a fibrous capsule, frequently with an irregular polycystic surface. On cut section the multiple cystic elements are again identified. Usually the cysts are lined by a single layer of low cuboidal epithelium supported by a moder-

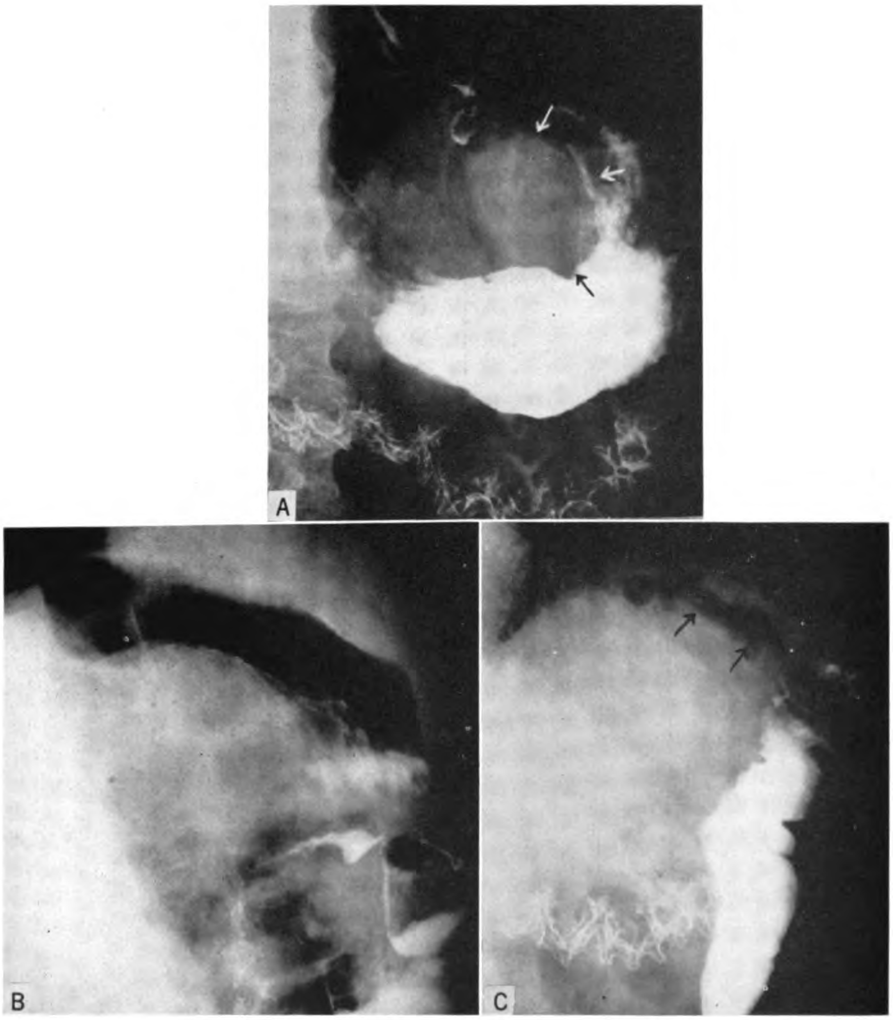

FIG. 104.—*A*, Carcinoma of the body of the pancreas in a 63-year-old man with weight loss. No definite pain. Supine roentgenogram of stomach shows indentation into lesser curvature (arrows).

B, Same patient, lateral view after ingestion of soda water. The tumor indents deeply into the posterior wall of the pars media.

C, Same patient, post-evacuation film of barium enema. The splenic flexure is displaced laterally by the mass. The stomach gas bubble also is displaced (arrows).

ately hyalinized or partially calcified stroma. Calcification within the cyst wall often can be identified on scout abdominal roentgenograms.

Benign pancreatic cysts, whether of developmental or degenerative origin, present a somewhat characteristic picture. The patient complains of a dull achy, colicky pain, usually in the epigastrium or left hypochondrium, sometimes radiating to the back. If the mass is sufficiently large it can be palpated. These cysts vary in size from about 2 to 3 cm. in diameter to well over 25 cm.

The presence of a circumscribed opacity on scout abdominal roentgenograms, often with calcium in its periphery, should suggest the possibility of a pancreatic cyst. If the cyst is in the head of the pancreas, its pressure produces a diffusely widened and relatively smooth duodenal loop, the straight-

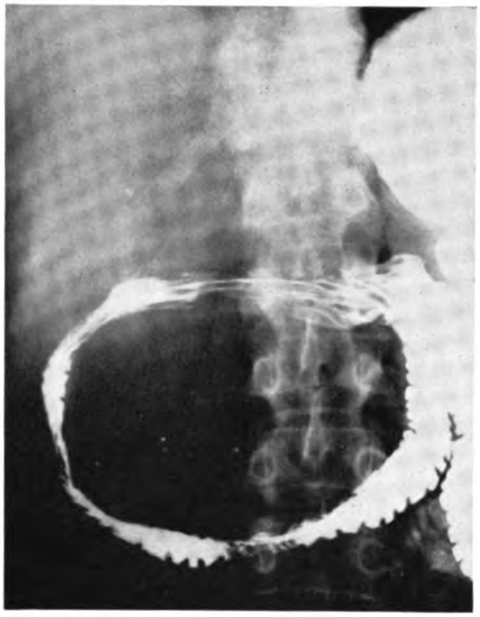

FIG. 105.—Pancreatic cyst in a 68-year-old woman with back pain and slowly increasing jaundice. The mass had been present for 3 years. At operation the head of the pancreas contained a cyst about 12 cm. in diameter. Serosanguineous fluid was aspirated and the cyst extraperitonealized. Malignant tissue was found in its wall.

ened impression along the medial aspect of the loop corresponding with the periphery of the cyst. The cyst also can impinge on the inferior aspect of the pylorus in a smooth curvilinear fashion, and extend downward against the transverse colon as well. Cysts in the body of the pancreas impinge against the lesser curvature of the stomach, and are better identified on lateral films in some cases. Cysts of the tail of the pancreas lie close to the cardia, and in 2 cases seen by us simulated carcinoma at the cardio-esophageal junction. In 1 case definite dysphagia was present. If calcified, these cysts may be confused with aneurysms of the abdominal aorta or splenic artery, as occurred in 2 of our patients.

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TUMORS OF THE SPLEEN

Usually the clinical picture is decisive in determining the cause of splenomegaly. The differential diagnosis includes leukemia, other blood disorders such as congenital hemolytic anemia or erythroblastic anemia or lymphoma. Sometimes Gaucher's disease presents itself first with diffuse splenomegaly.

Rarely the spleen may contain a local tumefaction as part of the picture of Gaucher's disease. In one of our cases, a 58-year-old man, the radiologic appearance suggested a splenic cyst. On inspecting the removed spleen, the pathologist believed that we had an unusual hemangioma. The histologic examination, however, disclosed a classical picture of Gaucher's disease throughout the spleen, in addition to a mass of circumscribed involvement which grossly simulated a hemangioma. Cysts of the spleen are relatively uncommon, and often are of congenital origin. Echinococcus cysts also occur, usually as multiple structures.

Occasionally a primary sarcoma originates in the capsule of the spleen. The outstanding characteristic of this condition, as noted in 6 cases seen by us, was a diffuse adhesive process to adjacent structures. Spindle cell sarcomas are rare, occurring as either small or bulky tumors. Endothelial sarcomas occur as multiple nodules in a greatly enlarged organ.

Extension of tumors such as gastric or colon carcinomas into the spleen are accompanied by considerable splenomegaly. Rarely a retroperitoneal tumor invades the spleen, or displaces it downward by contiguity.

Other diseases in which splenomegaly is prominent include infectious mononucleosis, cirrhosis of the liver, and rarely splenic abscess.

The radiologic diagnosis of splenic tumor is predicated on the identification of diffuse enlargement, or localized cyst-like alterations in contour. The adjacent viscera may be displaced, the stomach to the right and anteriorly, and the splenic flexure downward and anteriorly. The left kidney may be displaced downward if that portion of the spleen between the diaphragm and stomach is enlarged. However, if the splenic enlargement is such that its anterior aspect mainly is affected, the kidney may remain in its normal position. In a review of 60 of our cases of pronounced splenic enlargement, downward displacement of the left kidney was found in only 35.

As a rule splenomegaly does not interfere with the motion of the left diaphragm except when it is so bulky that the mechanical effect prevents excursion. Even with pronounced splenic enlargement the diaphragm is not elevated, and pleuropulmonary irritation is uncommon. Rarely a gastric neoplasm invades the spleen, producing a fistulous tract. We saw one such instance in a 46-year-old-man in whom the diagnosis was confirmed at operation.

Accessory spleens should be considered as a congenital variation. Once in a while, however, the spleen is displaced on a long pedicle, thereby simulating an upper abdominal mass. Rarely, the spleen is displaced into the lower abdomen, and appears as a pelvic tumor. In one of our patients, a 45-year-old woman, the preoperative diagnosis of an ovarian tumor was changed to ectopic spleen. The spleen was found on a long pedicle in the right lower quadrant. It weighed 700 grams, and no cause for its enlargement could be identified.

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TUMORS OF THE PERITONEUM

Primary tumors of the peritoneum are uncommon. Occasionally a sarcoma of the omentum is found, but this diagnosis is usually made at operation. This lesion escapes notice until it either becomes large enough to be detected on physical examination, or the patient develops diffuse abdominal complaints and ascites. Radiologically, they are sometimes identified as relatively well-circumscribed masses, but more often they are obscured by intra-abdominal fluid. Sometimes pressure effects on adjacent bowel indicates the presence of a mass. These tumors extend locally to the serosa of the small intestine, producing partial intestinal obstruction with recurrent abdominal distress and pain. In 1 case seen by us, a 45-year-old woman, the clinical diagnosis was twisted ovarian cyst. At operation a fibrosarcoma originating near the jejuno-ileal area was found which measured about 50

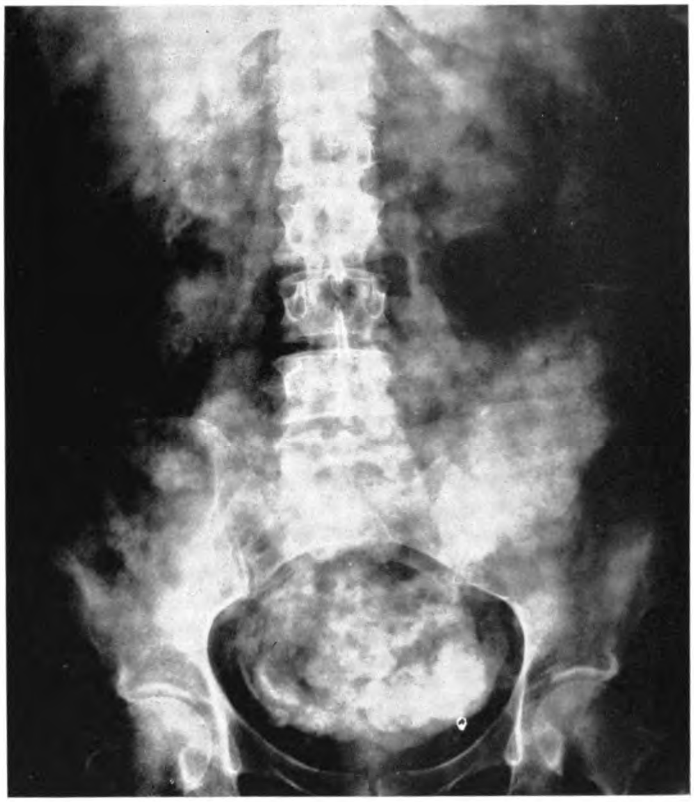

FIG. 106.—Psammomatous peritoneal calcification of ovarian metastases in a 55-year-old woman, verified at operation.

cm. in diameter. This patient had had a 2-year history of intermittent abdominal pain with a gradually increasing mass in the lower abdomen. A second case was that of a 72-year-old woman admitted because of progressive increase in the size of the abdomen attributed to ascites. Radiographic examination disclosed displacement of the colon. The diagnosis was ovarian carcinoma with peritoneal metastases. The true nature of the tumor was established at operation.

Another infrequent peritoneal tumor is a mesothelioma. This is seen in young people, and presents as a localized abdominal mass. If sufficiently large, it displaces the adjacent bowel. In these youngsters the preoperative diagnosis often is neuroblastoma.

Lipomas of the mesentery also are infrequent, and are sometimes identified by the radiolucent character of the fatty content. These occur as cystic or solid tumors, the former being more frequent. At times lipomas are large, and are associated with chronic complaints. When associated with incomplete rotation of the cecum and ascending colon, volvulus with acute obstruction can develop. There is no tendency for sarcomatous degeneration such as may be seen with retroperitoneal lipomas.

Dermoid cysts of the mesentery are usually found near the distal ileum, but occur elsewhere also. These appear as sharply-delineated masses of varying size, often containing numerous calcifications. Symptoms are due to complications such as bowel obstruction by kinking, volvulus, intussusception or torsion and vascular impairment. Peritonitis or hemorrhage follow sloughing off of a blood vessel. Confusion with ovarian dermoids is not infrequent because of the similarity between these lesions, particularly when torsion occurs. A rather pronounced ileus often develops with torsion of a cyst.

Another unusual form of cystic disease of

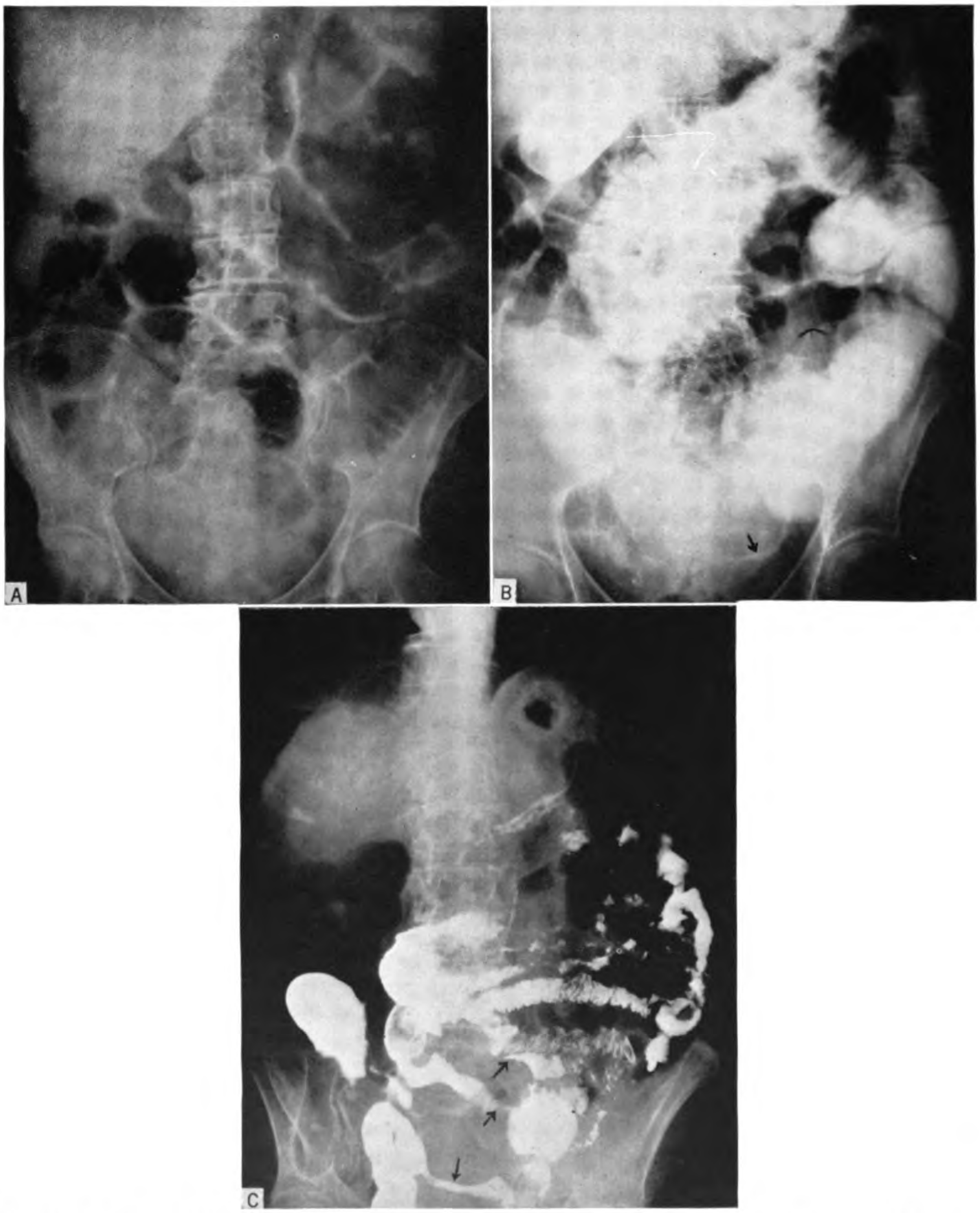

FIG. 107.—*A*, Ileus due to peritoneal carcinosis from a sarcoma of the omentum in a 56-year-old woman. The lesser bowel is diffusely dilated.

B, Three hour film after 90 c. cm. of 70 per cent Hypaque by mouth. The contrast medium is still in the small intestine. A constricted area of ileum is present in the pelvis (arrows).

C, Same patient, 3 hours after a barium meal. Scattered areas of constriction of the ileum are present These were produced by serosal implants of tumor, causing partial intestinal obstruction (arrows).

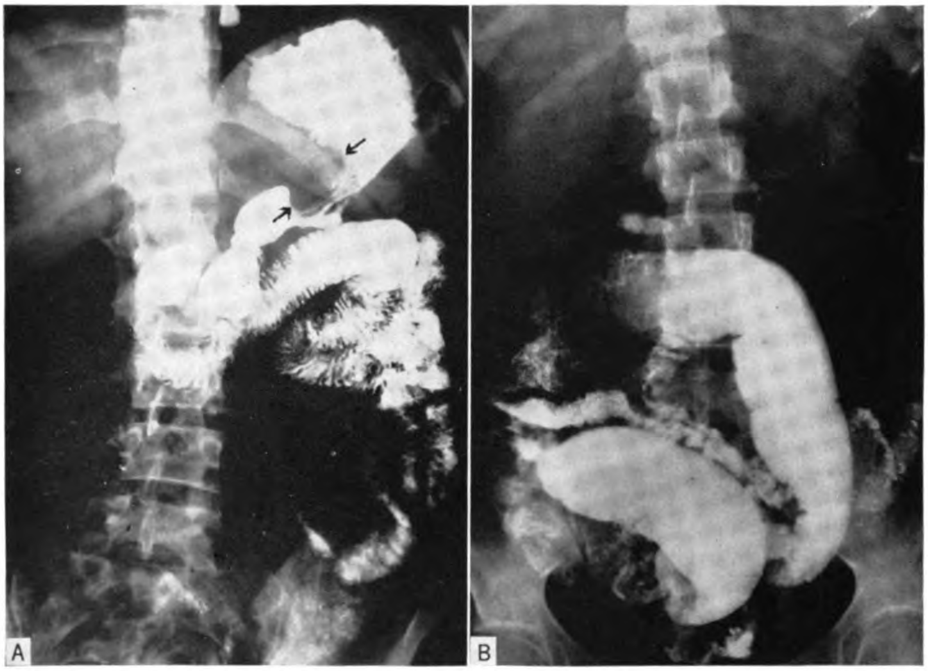

FIG. 108.—*A*, Carcinoma of the stomach (arrows) in a 48-year-old woman with intermittent diarrhea, constipation and abdominal distention.

B, Same patient, 5 hours later. A distended loop of bowel is seen in the lower mid-abdomen. This was found to be a markedly distended ileum, obstructed in the right lower quadrant by a dense metastatic mass imbedded around the bowel.

the peritoneal cavity is encountered with Kaposi's sarcoma. A unique case with numerous intra-abdominal cysts was described by Sherwin and Gordimer. They reported cysts which attained the size of an infant's head, one of which had ruptured into the small intestine and appeared as a large air-containing cyst.

The most frequent peritoneal neoplastic disease is that due to metastases. While these are frequent with ovarian carcinoma, similar changes appear with other cancers such as those of the gastrointestinal tract, the pancreas, the cervix and breast. No characteristic sign is associated with peritoneal carcinosis. Not infrequently these patients have no symptoms referrable to the original neoplasm, but are seen because of diffuse abdominal distress and later ascites and partial intestinal obstruction.

Peritoneal carcinomatous implants occasionally stud the entire abdomen with tumors of varying size. These deposits may cause ascites, partial obstruction and ileus. Those originating from ovarian carcinomas are sometimes accompanied with a peculiar psammomatous calcification within the metastases, whether they be peritoneal, omental or parenchymatous. Single deposits in the serosa may deform the bowel, simulating colonic neoplasm, or if localized, sessile polyps. Occasionally, bowel loops become matted and adherent, and fistula formation occurs as a futher complication.

Radiographic examination of patients suspected of intestinal obstruction due to peritoneal carcinosis can be facilitated by preliminary study using water soluble nonabsorbable contrast material. If this passes freely, it is then safe to proceed with a definitive study with barium. There is considerable difference of opinion as to whether or not one might properly start the study with barium, it being held that only partial obstruction permits barium to pass without difficulty, while complete obstruction requires operation nevertheless.

The radiographic appearance of ascites

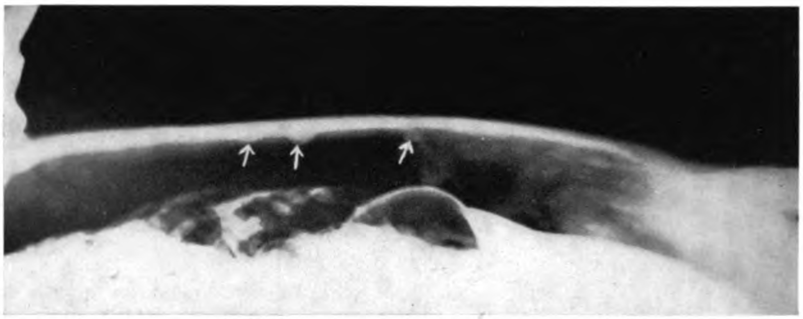

FIG. 109.—Trans-abdominal view of artificial pneumoperitoneum reveals metastatic tumor nodules on parietal peritoneum (arrows).

with peritoneal carcinosis does not differ from that of other causes. A diffuse haze in the film appears over the abdomen when sufficient fluid accumulates. Occasionally fluid becomes encysted, producing a localized density, the nature of which usually is determined at operation.

Neoplasms of the intestinal tract perforate because of degeneration or trauma incident to examination of the colon. In either event, pneumoperitoneum is produced with the usual signs of free air in the peritoneal cavity. If perforation occurs during barium enema, a peculiar gathering of the contrast material along the periphery of the abdominal cavity appears as a first and almost immediate indication.

Implants or direct extension of cervical cancers may impair the patency of the ureters, producing slowly progressive hydroureteronephrosis. This often is associated with a pelvic mass sometimes identified radiologically by virtue of its bulk or by its effect on adjacent viscera, particularly the colon and bladder. Bladder and rectal tumors also perforate into the pelvic peritoneal cavity and cause masses whose nature are determined from the history as well as from the changes due to such disruption of continuity.

Endometriosis with serosal implants causes filling defects in the colon simulating intrinsic disease. As a rule, this diagnosis is suggested clinically. Occasionally these lesions affect the small intestine and cause acute obstruction.

There is no radiologic way to identify a Bloomer's shelf. This condition, so readily diagnosed by digital rectal examination, is not apparent on abdominal roentgenograms unless pressure effects are exerted against adjacent structures.

Ascites sometimes causes protrusion of the umbilicus. The presence of neoplastic nodules in such hernias should be checked for by palpation. In one of our patients, radiographic study of the protruding umbilicus disclosed small calcifications identified as psammomatous ovarian carcinoma metastases.

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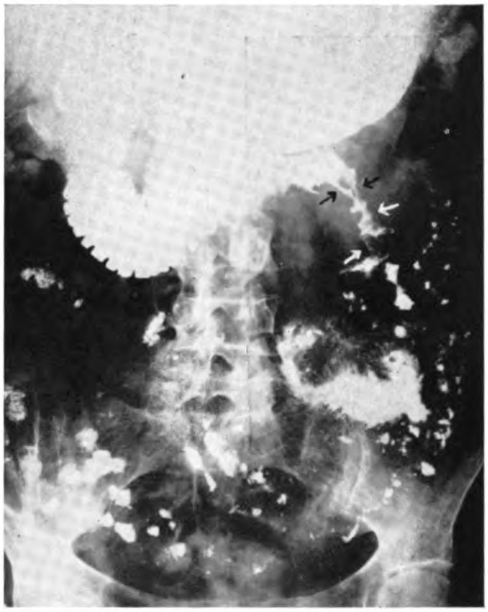

FIG. 110.—Retroperitoneal lymphoma in a 69-year-old man with weight loss and intermittent vomiting for 6 months. Five-hour film shows marked gastric retention with duodenal dilatation. The infiltrated area in the jejunum was due to extension from the root of the mesentery (arrows).

RETROPERITONEAL TUMORS

The most frequent malignant retroperitoneal tumors are lymphomas. Hodgkin's disease, reticulum cell sarcoma and tumors with leukemic origins occur either as isolated masses or diffuse tumorous invasions. These may be asymptomatic, particularly in their early stages. However, as they increase in size, a gradually progressive chain of vague symptoms appear. At first nothing more than anorexia, abdominal distress, pain and sometimes vomiting are noted. Secondary anemia, weight loss for no apparent reason and weakness are common. If the tumor invades the diaphragm, pain is noted in the corresponding shoulder. Later these patients become distended, at times so much so that the diagnosis of intestinal obstruction is made. With invasion of the abdomen, the tumor proceeds into the posterior surfaces of the lesser or large bowel, causing hematemesis and gastrointestinal bleeding. Occasionally jaundice follows extension into the porta hepatis.

The radiologic manifestations of retroperitoneal tumors reflect its size and extension. Small lesions produce no visible changes. Later the psoas muscle on the affected side becomes indistinct, and displacement of adjacent viscera follows, depending on the size and location of the mass. An interesting group of these cases is that in which the tumors are located near the duodenal sweep, simulating tumors of the head of the pancreas. Displacement of the kidneys occurs with growths high in the retroperitoneal areas. Here the kidneys can be displaced laterally, and either upward or downward. With tumors lower in position, the ureters are displaced and compressed laterally or anteriorly, and occasionally there may be depression of the bladder.

The psoas shadows are of particular interest. Our experience has been that diminution of their sharpness is not a reliable sign of retroperitoneal tumor. In a review of 40 verified cases, only 6 showed definite abnormalities of these structures. In 7 other patients, there was a fairly definite increase in the density of the ipsilateral psoas shadow, and in 5 of these a lateral outward bulging of the muscle was present.

Recently in 3 patients who had concomitant calcification of the abdominal aorta, a definite forward displacement of this structure was present, indicating retroperitoneal tumor involving the retroaortic region.

Extension of lymphomas into the mesenteric root may produce changes in the configuration of the lesser bowel. Occasionally it is difficult to differentiate an intrinsic bowel lesion from a retroperitoneal lymphoma extending into the root of the mesentery.

Occasionally necrotization within a retroperitoneal tumor results in perforation into the abdominal cavity and the gastrointestinal tract. Invasion of the omentum and the peritoneum is associated with ascites and intra-abdominal masses, with the consequent clinical manifestations.

Retroperitoneal metastases appear from various neoplasms, those of the prostate, testicle and colon being fairly common. The radiologic manifestation of such extension is much the same as noted with primary retroperitoneal tumors, except that in our experience extension of such metastases into

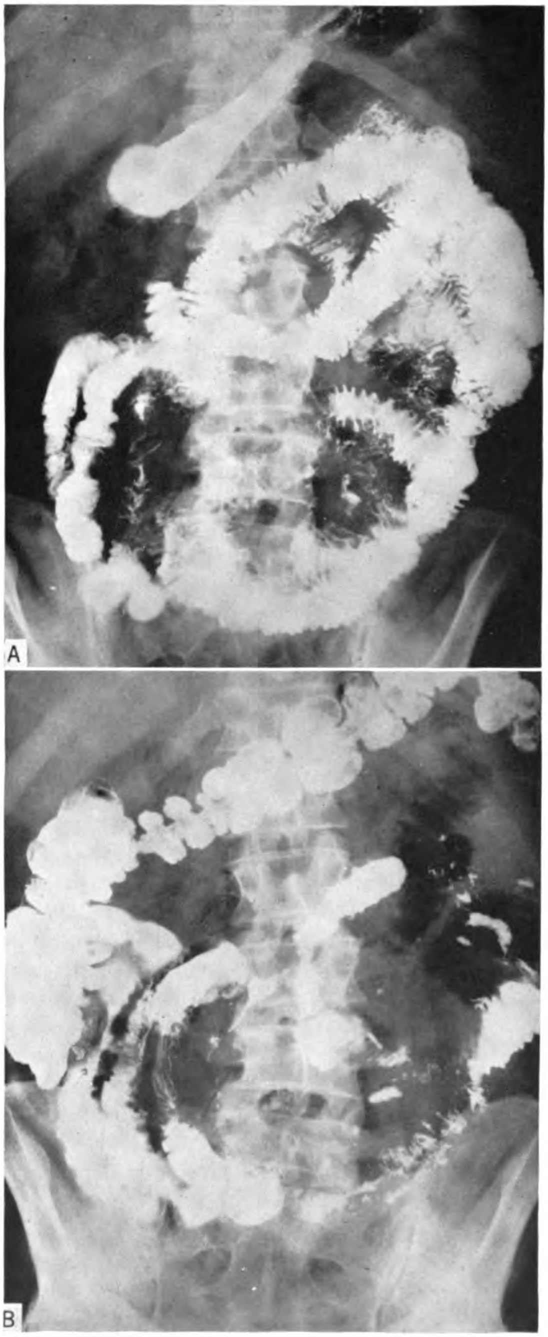

Fig. 111.—*A*, Retroperitoneal lymphosarcoma in a 56-year-old male with cramps and vomiting for 6 weeks. One hour film shows hypomotility, dilatation of small intestine and coarsened rugal folds.

B, Same patient, 6 hour film. The terminal ileum shows some narrowed areas (arrows) and considerable spasm. Note the resemblance to a malabsorption pattern or regional enteritis.

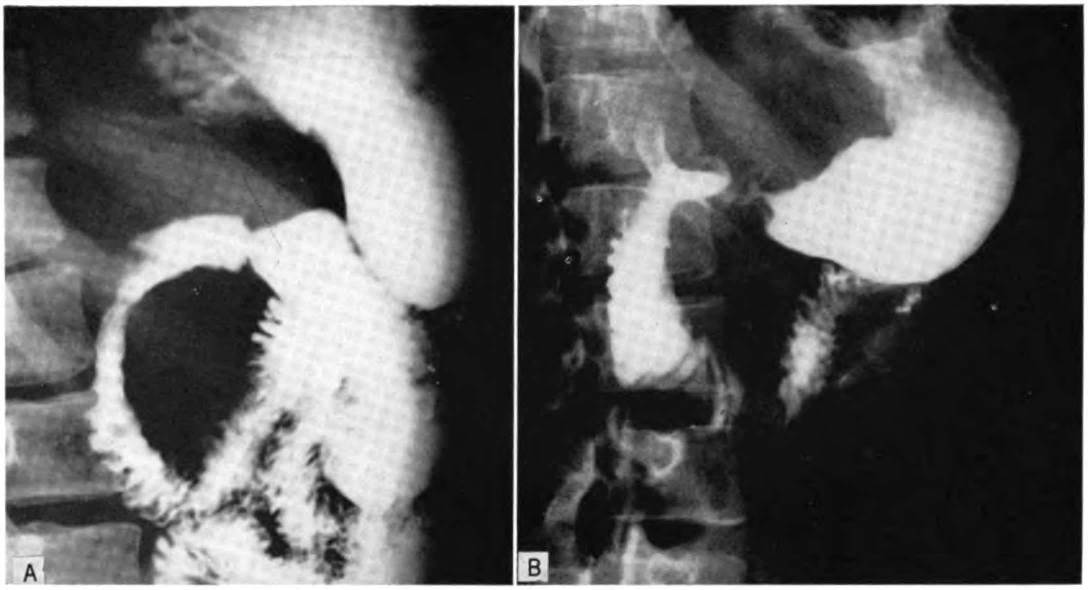

FIG. 112.—*A*, Reticulum cells arcoma causing widening of duodenal loop in a 17-year-old boy with nausea, vomiting, weakness and jaundice. This receded after *x*-ray therapy.

B, About 5 months later severe gastric bleeding appeared during intensive steroid therapy. This roentgenogram was made after patient swallowed 90 c. cm. of 90 per cent Hypaque solution. An ulcer is present on the distal aspect of the lesser curvature. The duodenal sweep is smaller than seen on the preceding film. At operation a bleeding ulcer was found. The region of the porta hepatis contained neither demonstrably enlarged lymph nodes nor tumor.

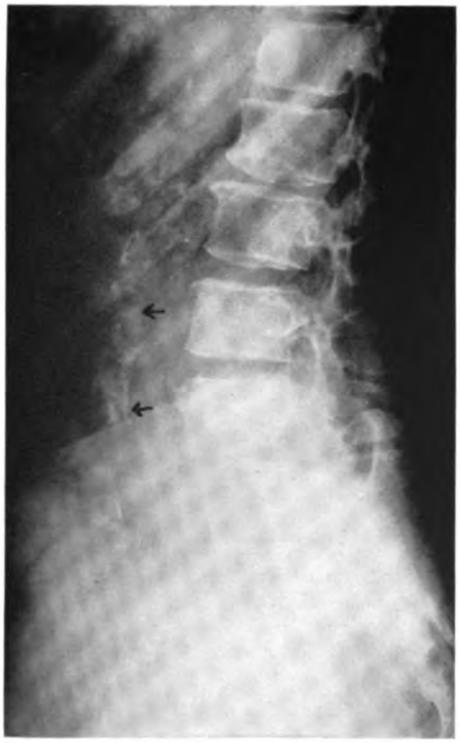

FIG. 113.—Forward displacement of calcified abdominal aorta (arrows) by retroperitoneal metastases from carcinoma of the prostate, verified at postmortem.

the peritoneal cavity and mesentery is uncommon.

Retroperitoneal tumors in infancy and childhood are usually neuroblastomas, renal embryomas and teratomas. These appear as palpable masses. Radiologically they produce distortions of the adjacent viscera. The nature of these lesions is sometimes revealed by calcium or formed elements within the mass. Teratomas are usually seen in the first 2 years of life, but occur later as well. While seen most often in the sacro-coccygeal area, they also appear in the retroperitoneal paravertebral gutter. Teratomas may undergo malignant degeneration.

The occurrence of retroperitoneal cavernous hemangioma associated with skin hemangiomas in a newborn infant was mentioned by Aurelius, Peterson and Niknejad.

The occurrence of retroperitoneal cysts, some containing chyle, should be mentioned here. Their etiology is believed to be lymphatic duct obstruction or effusion of chyle into a preformed cyst. They occur at any age, and while most common in the mesentery and omentum, occur retroperitoneally as well. The cysts are asymptomatic, unless torsion occurs, or traumatic rupture or hemorrhage cause acute symptoms. Occa-

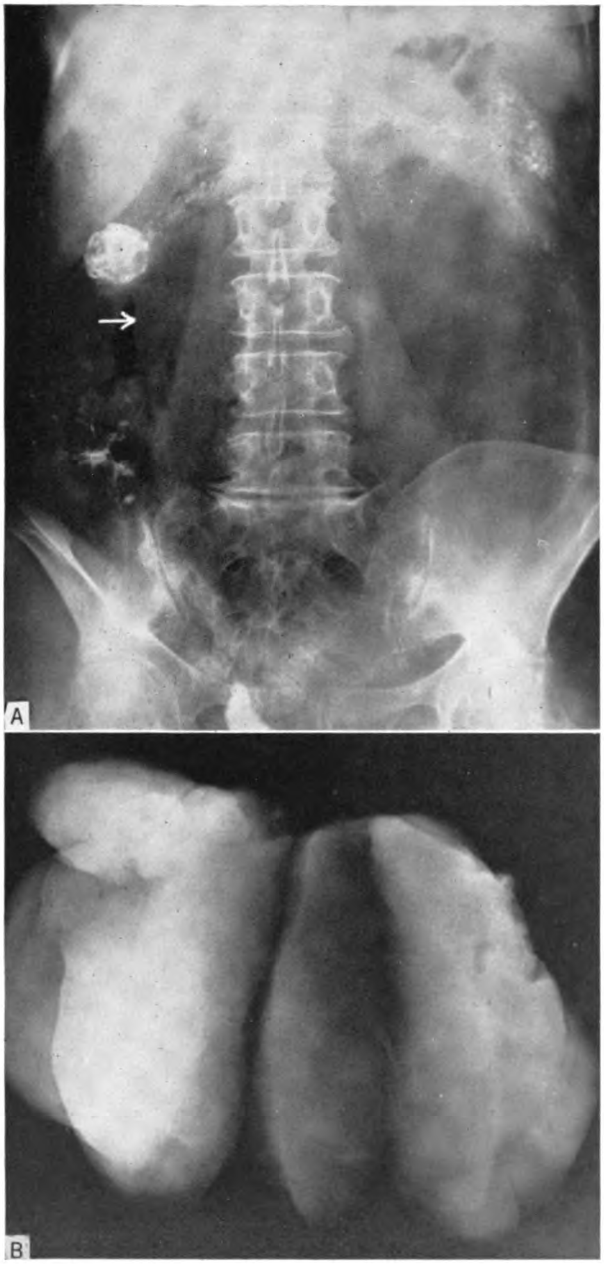

FIG. 114.—*A*, Radiolucent retroperitoneal lipoma in a 63-year-old woman with a large, increasing abdominal mass (arrows). Post-evacuation film, barium enema, shows displaced colon.
B, Radiogram of cut specimen.

sionally such cysts become large, as reported by Simon and Williamson in a 68-year-old man who had a retroperitoneal chylous cyst which practically filled the right side of the abdomen.

The presence of unilateral multicystic disease of the kidney in newborn infants is of interest. This is sometimes identified on physical examination, often associated with other urinary tract malformations. Direct

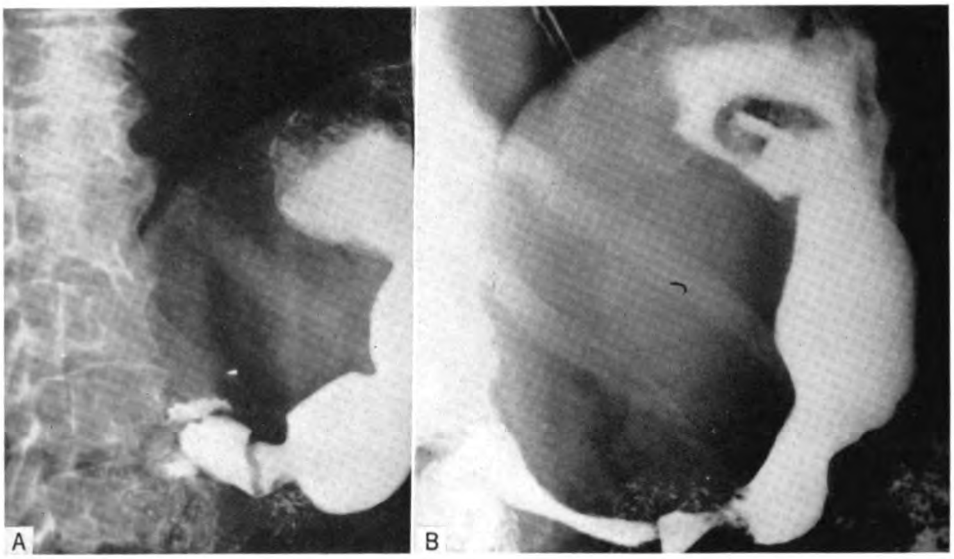

FIG. 115.—*A*, Retroperitoneal fibrosarcoma in a 55-year-old woman with anemia, weakness and rapidly increasing epigastric mass. Right oblique film shows no definite deformity of stomach.

B, Same patient 3 months later. A large mass impinges against the posterior wall. At necropsy the mass was adherent to the spleen, liver and stomach, and invaded the lesser omental sac.

examination of the abdomen discloses a soft tissue mass displacing the abdominal viscera. Urography disclosed a nonfunctioning kidney, but retrograde examination reveals distortion and dilatation of the involved urinary tract.

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Chapter 3

Inflammatory Disorders; Parasitic and Granulomatous Diseases; Collagen Disturbances; Metabolic Disturbances; Vascular Disorders

GASTRITIS

GASTRITIS often is superimposed on benign hypertrophy of the gastric mucosa, and also is observed with mucosal atrophy or any exudative change. All three processes can exist simultaneously. The condition is seen fairly often in young adult males, and in some is associated with mild ulcer-like symptoms. Gastric bleeding varying from occult blood to violent hemorrhage is of relatively frequent occurrence.

The distribution of thickened rugal folds with gastritis follows the normal pattern. There may be considerable stiffness, or the rugæ may be movable, changing their configuration with gastric activity, or when manipulated by manual pressure as seen under fluoroscopic control. The inflammation superimposed on thickened rugæ often extends quite deeply into the submucosa. The inflammation may be relatively localized to part of the stomach, or may involve discrete areas with the formation of grape-like masses which at times go on to the formation of pedunculated polyps.

Great caution is required in the evaluation of hypertrophic gastritis. Many times this is purely incidental, and has no clinical importance. When extensive and associated with localized rigidity, it cannot be accurately separated from malignant disease, and at times histologic confirmation is required before the diagnosis is established. Indeed, an etiologic relationship between the two conditions has been suggested. Texter and his co-workers observed 6 patients with gastric cancer out of 220 cases of gastritis. One patient followed for 14 years finally was operated upon because of melena, even though there was no radiologic evidence of cancer. A mass 10 cm. in diameter was found in the upper third of the stomach. The resected specimen revealed an area of ulcerated hypertrophic gastritis. A firm nodule of bizarre malignant glandular tissue was found in the margin, and the patient expired a year later with extensive metastases. The similarity between superficial spreading gastric carcinoma with rugal hypertrophy was described by Golden and Stout.

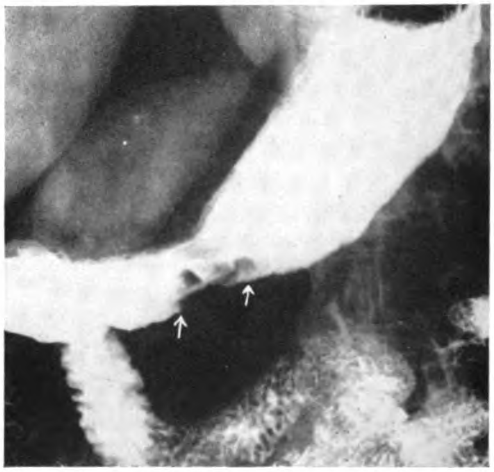

FIG. 116.—Localized hypertrophic gastritis in a 53-year-old man with vague upper abdominal symptoms. The irregular defect in the midportion of the greater curvature (arrows) disappeared within 6 months.

Localized hypertrophic gastritis presents an even more formidable diagnostic problem. Patches of thickened rugæ along the lesser curvature may simulate malignancy so well that operation often is unavoidable. Similar lesions along the greater curvature are some-

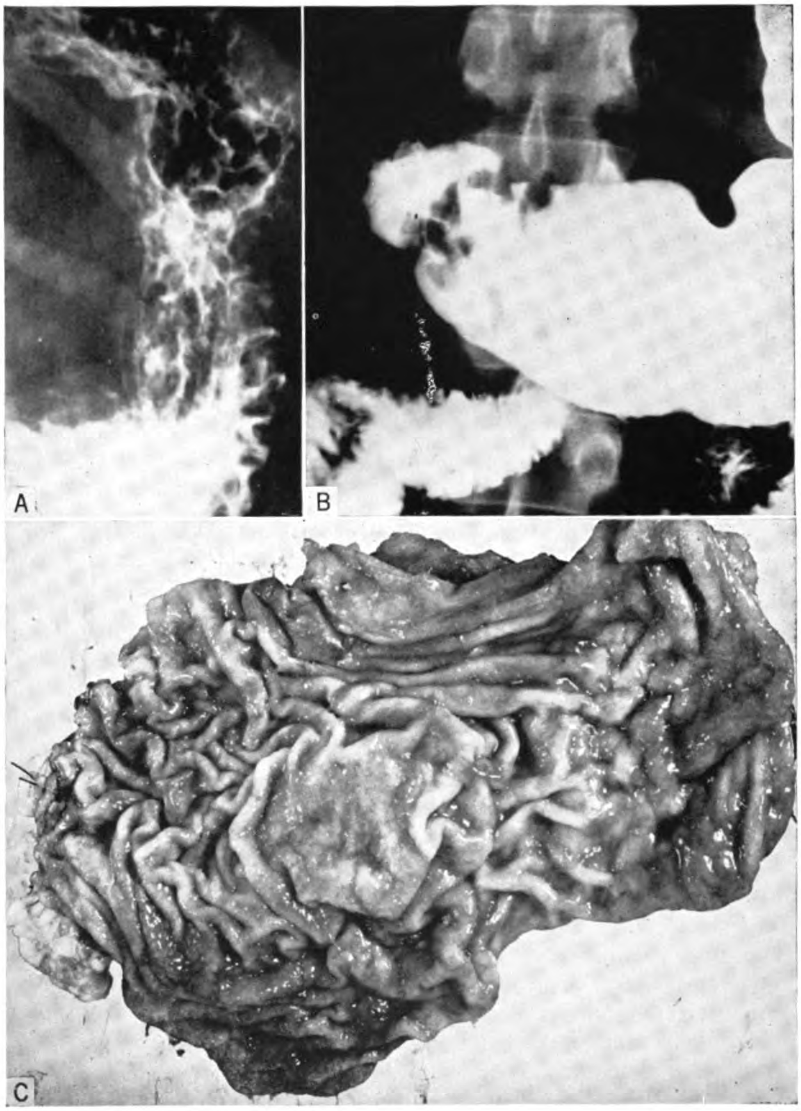

FIG. 117.—*A*, Spot roentgenogram in a 47-year-old woman with intermittent mid-abdominal pain, epigastric distress and loss of appetite. The rugal folds are diffusely thickened.

B, Same patient, posteroanterior supine stomach roentgenogram. Note the thickened antral rugæ.

C, Operative specimen reveals diffuse hypertrophic gastritis and antral gastritis. No ulcer crater could be found.

what easier to evaluate. Difficulty is heightened when localized patches of hypertrophied mucous membrane protrude well into the lumen, simulating polypoid tumors.

Gastritis also occurs acutely. Among the inciting conditions are excessive use of alcohol or tobacco, disturbances of mucosal function by physical factors such as chemicals and medications, or allergic changes. Acute gastric mucosal edema due to dietary indiscretions or ingestion of irritating substances causes transient thickening of the rugæ which disappears within a few days. Sometimes the folds are coated with excessive mucous which prevents the contrast material from adhering. A moderate atony and

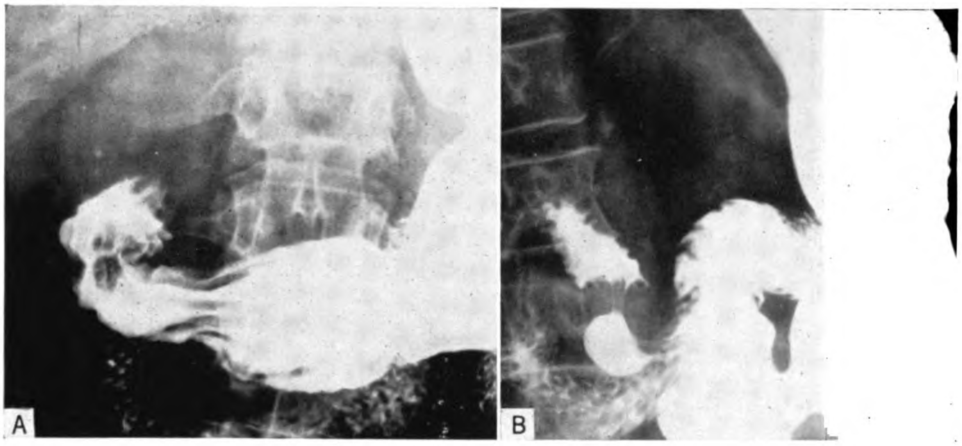

FIG. 118.—*A*, Protrusion of antral mucous membrane into base of duodenal bulb simulating polyps. Patient is a 55-year-old woman with vague upper abdominal pain. Previous study 7 months ago was identical with the present examination.

B, Same patient, right oblique prone view showing normal pyloroduodenal segment.

poor peristalsis also appears with this condition.

Antral gastritis presents a wide variety of mucosal changes and clinical symptoms. In some patients there may be no subjective manifestations of the thickening of the rugal folds at the antrum, while in others symptoms of peptic ulcer are prominent. The mucosal patterns at times show nothing more than fairly symmetric thickening of the rugal folds, which are quite flexible and movable. In others, there is marked distortion of the mucosal pattern, together with irritability and spasm which not infrequently is associated with antral deformity indistinguishable from malignant infiltration. The passage of barium through the antrum usually is not grossly impaired, although moderate retention after 5 hours is not uncommon. Gastric dilatation due to obstruction is rare. Concomitant pyloric ulcers are rather frequent. These may be small and superficial, requiring careful search of the specimen before they are found. In a review of 80 of our verified cases, only 10 were free from ulcer. However, the radiologic identification of ulcer niches in this group was considerably less than anticipated. In many the niche was small and hidden by irregularly distributed hypertrophied and inflamed folds. Since the rugal folds can slide over the muscularis, it is not uncommon for them to bulge against the base of the duodenal bulb producing an arcuate indentation. The rugal folds also can pass through the canal into the duodenal bulb. With adequate compression studies, this condition often is properly identified. However, in some instances intraduodenal polypoid lesions are simulated. This is far more frequent with hypertrophied rugal folds, but also has been observed with atrophied gastric mucous membrane.

Transpyloric passage of antral mucosa occurs in young as well as in older people. Its presence in children is uncommon but we have seen several such cases, the youngest being a 4-year-old boy.

There is still much discussion as to the clinical import of prolapse of gastric mucosa into the duodenum. Some consider it a physiologic change not necessarily associated with symptoms. Others, however, ascribe symptoms such as epigastric pain, gas, fulness, belching, burning, nausea, vomiting and sometimes bleeding to this condition. It appears that this change is not necessarily a lesion associated with symptoms, and it is best not to attribute too much to it. It might be well to regard it as a complication of rugal prominence rather than a lesion producing symptoms on a mechanical basis. Levin and Felson observed that in 100 asymptomatic patients, gastric mucosal prolapse occurred in 18. The condition was slight in 10, moderate in 5, and marked in 3.

GASTRIC ABSCESSES

Localized abscess in the gastric wall usually are due to septic emboli, and cannot be diagnosed radiologically. They also form by direct extension from inflamed areas in the liver, spleen or pancreas. Gastric ulcers or carcinomas containing localized purulent collections are uncommon. In these the underlying process results in characteristic radiologic changes, and the abscess usually is unidentified unless air or fluid enter in sufficient quantity to arouse suspicion.

Diffuse phlegmonous infiltrations of the stomach wall are uncommon. This condition affects males in middle life most often. The infections usually are streptococcal, but pneumococci, staphylococci, colon bacilli and Welch bacilli all have been found. Severe physical exertion, alcholism and gastric hypoacidity are considered predisposing. Other causes mentioned are gastritis, gastric ulcer and carcinoma, recent gastric surgery, acute exanthematous infections and furunculosis. Phlegmonous gastritis caused by gas-forming organisms has been reported following attempted suicide with hydrochloric acid, and by penetration by contaminated foreign bodies such as fish bones.

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PHLEGMONOUS GASTRITIS

Pyogenic infection of the stomach wall usually begins in the submucosa and extends into the serosa, leaving the mucosa intact. If localized, the inflammation is confined to the submucosa, but if diffuse the stomach wall becomes thickened and the inflammation extends to and occasionally perforates the serosa. Inflammatory masses and gas vesicles accumulate between the muscularis mucosa and the submucosa. Scout abdominal films then disclose emphysematous gastritis with innumerable gas bubbles silhouetting the stomach. These bubbles gather along the gastric curvatures, particularly the fundus, and may extend up to the esophagus. They remain visible for some time, variously reported as from several days to a month. The condition rarely is diagnosed clinically, often being confused with peritonitis, acute pancreatitis, cholecystitis and nephrolithiasis. In its acute form the disease is sudden in onset, with severe pain, bloody vomiting, and a fulminating course leading to early peripheral collapse. The prognosis is poor, but recoveries have been reported after surgery or adequate medical treatment.

Phlegmonous gastritis also occurs in a chronic form, mimicking ulcer or carcinoma. The chronic stage ends in sclerotic scarring which cannot be distinguished from other cicatricial conditions.

In differential diagnosis of acute or chronic phlegmon of the stomach, the conditions to be considered are hyperplastic gastritis, ulcer, scirrhous carcinoma, lymphoma or granuloma. The presence of air bubbles in the stomach wall sometimes is observed with pneumatosis intestinalis also. The radiologic identification of the stomach encased in small bubbles of gas is of tremendous help in making the diagnosis.

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PEPTIC ULCERS

Peptic ulcers are infrequent in infancy Duodenal ulcers are less frequent than gastric ulcers up to the age of two years, and thereafter duodenal ulcers are more common. Peptic ulcers in infancy often are associated with brain damage and prematurity. Other conditions which predispose to this condition are bacterial infections with bacteremia, circulatory disturbances with mucosal anoxia and devitalization, and physical trauma and burns. Symptoms may be minimal and the diagnosis often is made at postmortem examination. Superficial ulcers predominate, and these are quite difficult to recognize radiologically. Vomiting in the newborn period with hemorrhage should alert the examiner to this possibility.

As a rule, the diagnosis of a perforated duodenal ulcer is first thought of when a spontaneous pneumoperitoneum occurs. In the absence of this, gastric ulcers are occasionally identified at the pyloric ring, the lesser curavature or the anterior wall of the stomach. As a rule no induration or cellular reaction is noted, although the more chronic ulcers have some peritoneal reaction about them. From the ages of 1 year to about 10, gastric ulcer is less frequent, and to a considerable extent is like that seen in adults both from the clinical and radiological viewpoint. In a recent review, Block found only 39 cases of true gastric ulcer in childhood, postulating that in acceptable cases symptoms must exist for about a month without concomitant predisposing conditions such as burns or infections. The importance of psychosomatic influences was commented upon by Goldsberry.

Gastric ulcers are frequent in adults. Their pathogenesis is one of a complex association of the proteolytic action of hydrochloric acid and hormonal influences. Ulcers range in extent from shallow mucosal excavations to complete erosion of the gastric wall, with perforation an ultimate outcome.

Acute gastric ulcers occur as complications of acute infections and central nervous system disorders, but it is not yet known whether the mechanism is the same as for peptic ulcers. Chronic peptic ulcers occur most frequently along the midportion of the lesser curvature, but are seen elsewhere as well. Those along the anterior and posterior walls, and high on the lesser curvature are rather uncommon, but are quite important when sources for gastric hemorrhage are sought. At one time all greater curvature ulcers were considered malignant, but repeated reports of benign ulcers in this location indicate that this is not invariable.

The clinical picture of gastric ulcer requires no elaboration here. Of interest are the acute aspects of the condition, such as hemorrhage, perforation or fistula formation. Massive hemorrhage from gastric ulcers reportedly is the cause of 10 per cent of such cases. With perforations of anteriorly situated ulcers, pneumoperitoneum with its attendant shock appears. Localized perforations sealed off by omentum or peritoneum prevents entrance of gastric contents into the abdominal cavity. These ulcers usually are situated at the lesser curvature and posterior wall, and by direct extension reach the pancreas, the liver, gallbladder, colon or subdiaphragmatic regions. Fistulæ from gastric ulcers to the colon have been recorded, and similar passages to the biliary tract also are known to occur.

Gastric ulcers are frequent in adults, and usually occur as relatively small lesions about 1 to 1.5 cm. in diameter. The gastric wall adjacent to the crater is indurated, and deformities appear with the development of serosal adhesions. Multiple ulcers are reported in from 5 to 10 per cent of cases. These are difficult to diagnose either clinically or radiologically. It has been suggested that symptoms of long duration with rare remissons might indicate this condition. The co-existence of gastric and duodenal ulcers is not uncommon, usually with the duodenal ulcer appearing first. In a group of 136 cases reported by Johnson, 64 per cent had pyloric stenosis and gastric retention, and this was regarded as favoring the development of gastric ulcers. These were found to be rather large and to bleed easily, and were more resistant to treatment than gastric ulcers unassociated with duodenal ulcers. It is difficult to postulate how rapidly a gastric ulcer forms, but evidence at hand suggests that they can heal quite speedily. Any ulcer which fails to heal within a month should be suspected of malignancy. The quickest disappearance time in our series is 10 days. The size of an ulcer cannot be correlated with malignant change. Giant ulcers over 8 cm. in diameter are seen rather frequently with no pathologic evidence of malignant degeneration. However, even from a purely anatomic viewpoint, it is often impossible to arrive at a definite diagnosis without histologic examination.

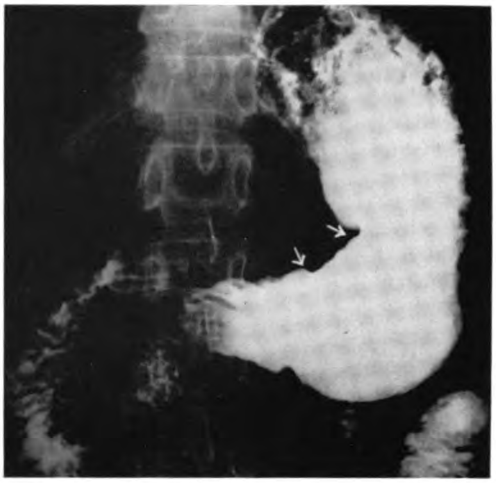

FIG. 119.—Small, flat inflammatory lesion in the midportion of the lesser curvature (arrows) in a 45-year-old woman with vague upper abdominal pain. The lesion persisted on two studies a month apart. At operation a localized area of inflammation with a tiny ulcer was found. Patient has remained well over the next 7 years.

The earliest gastric ulcers we have seen were associated with only a small area of rigidity along the midportion of the lesser curvature. In a few of these patients such fixation suggested the possibility of malignant infiltration, and the inflammatory nature of these lesions was established only after operation. No definite craters were identified in these patients.

Far more common is the smoothly outlined penetrating lesion surrounded by stiffened stomach wall. The adjacent mucosal folds sometime converge towards the ulcer, but more often maintain their normal positions.

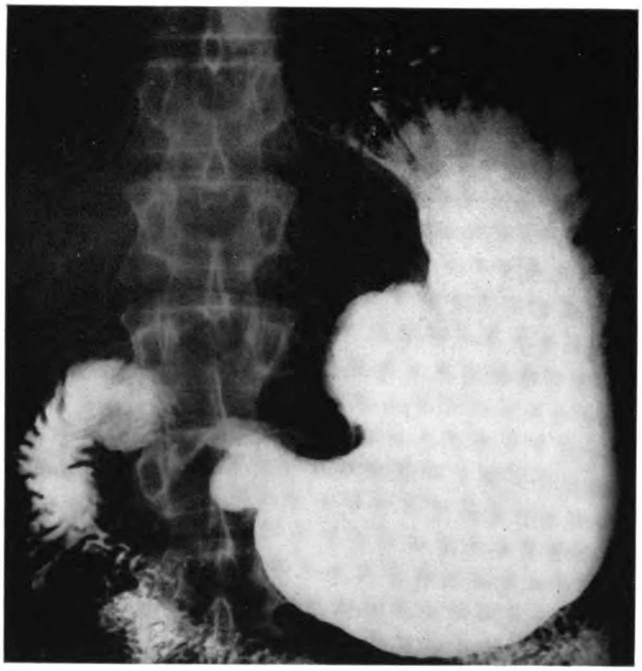

FIG. 120.—Large deep penetrating gastric ulcer in a 59-year-old man with long-standing abdominal distress. A 5-hour film revealed marked gastric retention. At operation a benign ulcer was found. No pyloroduodenal pathologic change was present to account for the gastric retention.

However, in anterior and posterior wall ulcers and in greater curvature ulcers there is more of a tendency for such convergence. Peristaltic activity in the ulcer vicinity often is diminished, but this is difficult to evaluate and as a rule is unreliable. The filling of an ulcer niche also is variable, the same defect at times being smaller or larger depending on the extent of filling and the angle of projection. Occasionally spasm of the wall opposite the ulcer produces an incisura, either at the same level or above it.

Differentiating benign from malignant gastric ulcers is a perplexing problem. Various signs have been enumerated, none entirely reliable. Projection of the niche beyond the stomach wall is believed to indicate a benign lesion, while failure to do so may indicate a malignant process. Radiation of mucosal folds towards the crater is considered a sign of a benign lesion, while irregularity of the niche and adjacent rugæ suggests a malignant change. A false sense of security may be obtained by slow healing of a gastric ulcer, inasmuch as malignant as well as benign ulcers present radiologic evidence of healing under adequate medical treatment, for a short time at least. Grimes and Bell suggested that proliferation of malignant tissue may obliterate a crater, thereby simulating healing. The question as to whether benign ulcers undergo malignant change still has not been settled. Marshall suggested that while the overall frequency of this changes is no more than about 6 per cent, the more practical problem was not to treat a malignant ulcer as a benign lesion. Of his 411 consecutive cases operated upon for gastric ulcer, 65 or 15.8 per cent were malignant, while of 346 benign ulcers 44 were considered malignant preoperatively. In 113 cases of benign ulcer, carcinoma could not be excluded. In contrast to this, only 1 of the 65 cases of proven malignant ulcer had a positive preoperative diagnosis, and in 37 carcinoma was considered as possible. An interesting observation was made by Comfort and his coworkers in a review of 326 cases of gastric carcinoma 4 cm. or less in diameter. They noted that the 5-year survivals were higher

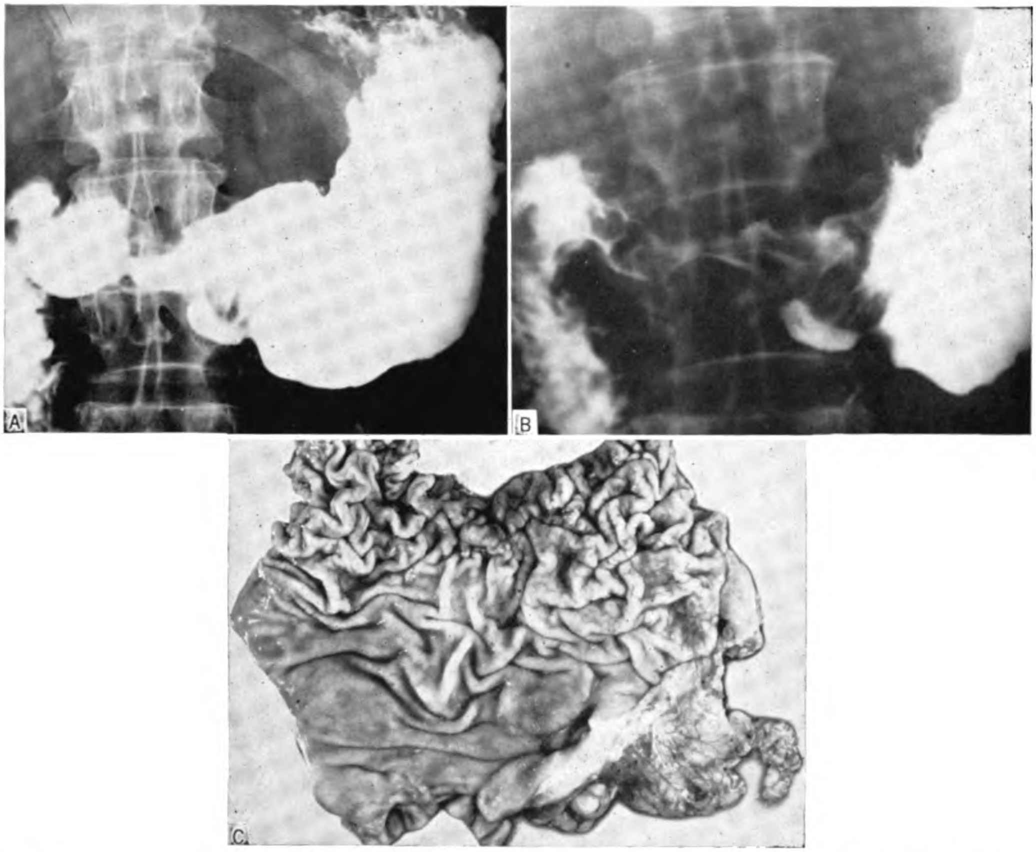

FIG. 121.—*A*, Benign gastric ulcer on the greater curvature of a 43-year-old man. *B*, Same patient, operative specimen. (Friedman, R. L. and Epstein, B. S., courtesy of Radiology, *55*, 398, 1950).

in patients with smaller lesions. They also found that these lesions tended to be less malignant and had a lower incidence of metastases, with a higher incidence of ulcer-like symptoms. Radiologic examinations were available in 222 cases. In 8, no gastric lesion was seen, while in the remaining 214 deformities were present. A radiologic diagnosis of carcinoma was made in about one-third of the cases, was suggested in another third, and in the remainder the roentgenograms did not suggest neoplasm. With lesions less than 1 cm. in diameter the correct diagnosis was made in 11.1 per cent of cases. With lesions 3 to 4 cm. in diameter the accuracy of diagnosis was up to 46 per cent. Swynnerton and Truelove surveyed 370 cases of simple gastric ulcer and carcinoma. In 26 there was evidence that the neoplasm

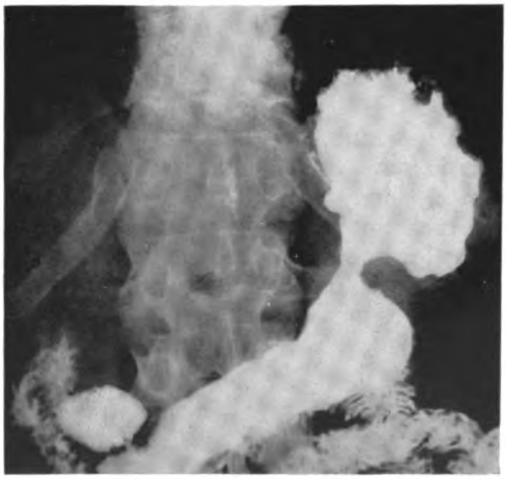

FIG. 122.—Gastric ulcer on lesser curvature of a 63-year-old man. A deep incisura is present on the greater curvature.

originated in the ulcer, 18 occurring in the prepyloric area and 8 in the body of the stomach.

In an analysis of 200 cases of massive upper gastrointestinal hemorrhage, Zimmerman noted that 22 had gastric ulcers. Of these, 3 died of the hemorrhage. The mortality of hemorrhage from gastric ulcer appears to be higher than from duodenal ulcer.

Gastrocolic fistula from benign gastric ulcer is uncommon. Gray stated that up to 1956 only 5 such cases had been reported. These tracts are identified better on barium enema examination, much the same as when a similar condition follows subtotal gastrectomy and gastroenterostomy. Spontaneous perforation of a greater curvature ulcer into the transverse colon was reported by Bosien and Tyson. Two cases of gastroduodenal fistula were reported by Bissylas, one secondary to an ulcerating gastric carcinoma and the second to a lesser curvature ulcer. In both patients the symptoms of abdominal distress were relieved somewhat after the fistula was established.

Gastric retention for more than 6 hours sometimes occurs in patients with gastric ulcer who have no organic change at the pyloroduodenal region to account for obstruction. It is believed that pylorospasm is the underlying factor. Nevertheless, great care should be exercised before assuming that no intrinsic disease is present. We have followed 6 cases in which no lesion to account for such gastric retention could be identified at operation.

Benign giant gastric ulcers present an atypical picture, with mild symptoms for years in some cases. These are seen more often in elderly patients. When associated with such symptoms as anorexia, pain and weight loss, a malignant lesion is somewhat more likely. One characteristic sometimes observed is persistent abdominal pain despite a rigid medical ulcer regimen. As a rule, these lesions appear on the lesser curvature and extend to the posterior wall, involving the middle and upper portions of the stomach. When they perforate into the adjacent retroperitoneal area, a large, hard indurated mass involving the stomach, pancreas, and in some cases the liver and mesentery is present. Often the nature of such a mass cannot be determined without histologic examination. Occasionally a large gastric ulcer is missed even with the most careful radiographic examination, and also escapes diagnosis by means of gastroscopy. The explanation for this is obscure, but it has been suggested that the ulcer sometimes is so large that it looks like a continuation of the stomach.

ANTRAL ULCERS

As with other peptic ulcers, antral ulcers start with mucosal edema and induration, followed by erosion, crater formation and scarring. The mucosal reaction to antral ulceration is quite intense, and antral gastritis of pronounced extent often accompanies small antral ulcers. Marked irritability and spasm of the antrum, together with thickening of the rugal folds are known to obscure antral ulcers, so that the lesion can only be identified if the rugal folds are separated and the crater is located. With periantral extension of inflammation, rigidity of the pylorus simulating neoplasm is produced. Duodenal irritability accompanying antral ulcers is frequent, resulting in bizarre configurations of the bulb. Conversely, patients with penetrating duodenal ulcers present with diffuse irritability of the antrum associated with periantral adhesions which may be so deforming as to overshadow the duodenal pathologic change and suggest that the primary disturbance is in the antrum.

Antral ulcers also occur as relatively discrete, small projections from the distal end of the lesser curvature of the stomach. This may be identified with difficulty in some cases because of overlapping of the antrum in certain oblique projections, and an obscuring effect is often produced by the proximal jejunum. It is important to obtain spot compression roentgenograms of this area early in the examination, preferably before the jejunum fills with barium. Accurate diagnosis of these lesions in some cases requires confirmatory studies.

Some antral ulcers extend to the pyloric ring and into the base of the duodenal bulb, so that considerable duodenal as well as

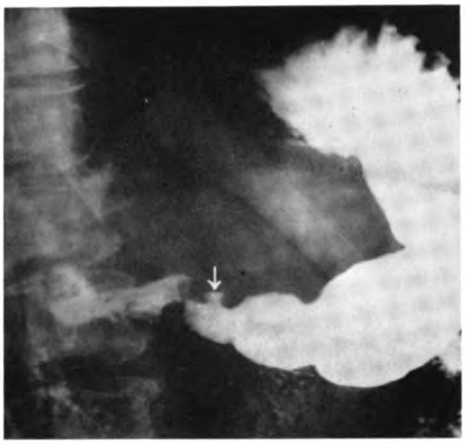

FIG. 123.—Benign penetrating ulcer at distal end of lesser curvature (arrow), verified as a benign lesion.

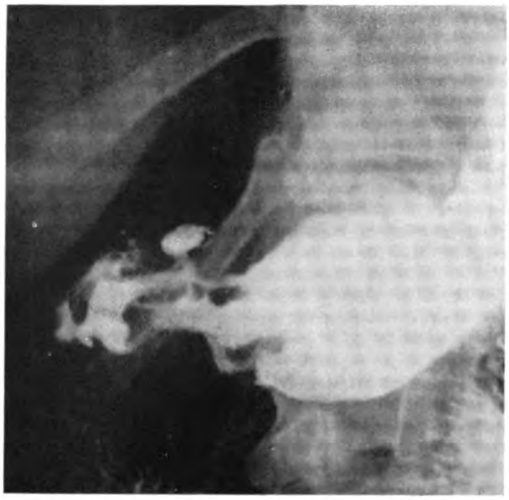

FIG. 124.—Benign penetrating antral ulcer with antral gastritis and peripyloric adhesions in a 48-year-old woman.

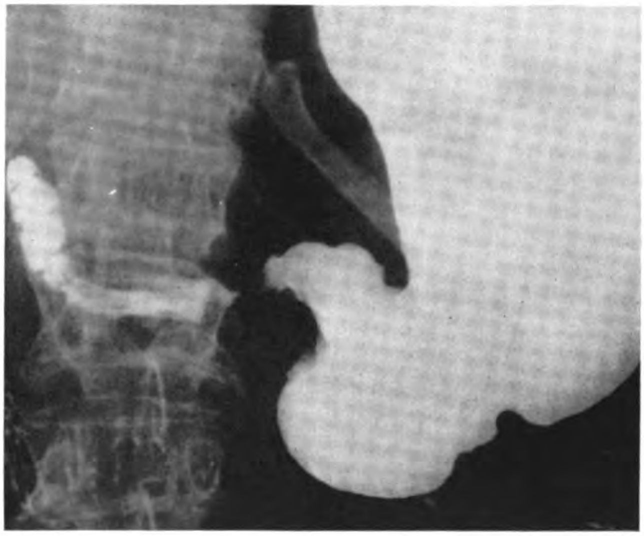

FIG. 125.—Pyloric obstruction due to chronic ulcer, with moderate 5-hour retention in a 67-year-old man. The ulcer crater is not visible. At operation a large prepyloric ulcer adherent to the pancreas was found.

antral deformity is produced. The identification of the point of origin of these ulcers is somewhat academic. Other radiologic changes seen with pyloric ulcers are lengthening of the pyloric canal and distortion and malfunction of this channel. Variable degrees of obstruction are encountered, from mild to pronounced gastric retention and dilatation. The latter is observed more often with cicatrization about the antrum, but significant gastric retention can be due to spasm as well. It is sometimes unwise to make a definite diagnosis of cicatrization purely on the basis of gastric retention, and a recheck several days later after the use of appropriate medical treatment is advisable.

A rather long history of vomiting food ingested several hours before points towards an organic obstruction.

Pyloric channel ulcers are relatively uncommon, the incidence reported by Boylston being about 0.5 per cent. Most patients are males in their fifth or sixth decade, and the clinical picture is not specific. Nausea, vomiting, upper abdominal pain and weight loss are frequent. Sometimes nocturnal periodic pain occurring before or after meals or with no relationship to food intake is prominent. It is believed that the pain associated with pyloric channel ulcers differs from the usual ulcer pain in being more colicky and constant.

Of Boylston's 26 cases, 1 was malignant. The average duration of symptoms was over 6 years. Accurate radiologic localization of the ulcer was obtained in 12 cases, the diagnosis being based on deformity of the base of the bulb, a poorly differentiated sphincter and pronounced antral spasm.

The advent of gastric retention because of pyloroduodenal cicatrization is followed by progressive gastric dilatation. Huber and Huntington brought out the point that gastric ulcers occasionally appear in the devitalized wall of such stomachs.

Traction diverticula of the stomach and duodenum are produced by fibrous bands attached to the serosa which exert an outward pull. This, like a benign ulcer, may appear as a pocket projecting beyond the gastric lumen. However, a tendency to be somewhat triangular in shape with a broad base and outward pointing apex is of diagnostic help. As a rule, these do not correspond in position with congenital diverticula, and the difference in configuration between these lesions is readily apparent in most cases.

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DUODENITIS

Diffuse inflammation of the duodenum presents a difficult problem to the radiologist and clinician. Symptoms which vaguely suggest a peptic ulcer are accompanied by an equally nondescript radiologic pattern. Duodenitis supposedly causes irritability, manifested by rapid passage of barium through the duodenum, retroperistalsis, and mucosal alterations varying from small stippled, irregularities to prominent thickening of the mucosal folds. An acute form of duodenitis sometimes is present with nonspecific gastroenteritis or other infectious diseases. Prevot reported a subacute variety of duodenitis and jejunitis following gastric surgery. The coexistence of duodenitis with duodenal ulcer has been observed frequently. It is interesting to reflect upon the frequency with which duodenal ulcer is seen, and the reluctance most observers have in making a definite diagnosis of duodenitis.

The differential diagnosis between a normal duodenum and duodenitis often is difficult. In the absence of a definite filling defect, distortion of the mucosal pattern sometimes has to be evaluated carefully for the possibility of ulcer. Another condition to be considered is hypertrophy of Brunner's glands. In this condition the nodulations in the duodenal mucosa are larger, and little irritability of the bulb is noted unless there are superimposed inflammatory changes. Diffuse lymphoid hyperplasia causes a somewhat similar picture

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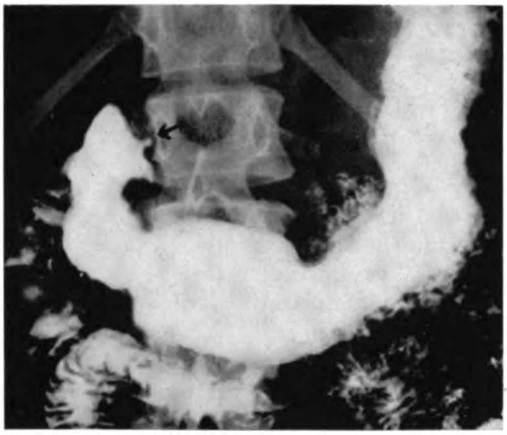

FIG. 126.—Acute penetrating duodenal ulcer on lesser curvature of the bulb (arrow) in a 47-year-old woman with melena and hematemesis.

DUODENAL ULCERS

Duodenal ulcers are the results of malfunction of the secretory and motor activities of the stomach. While the clinical picture is well known, the diagnosis nevertheless is often obscure, and the first signs are those of bleeding or the consequences of penetration or perforation. There is sufficient difficulty in diagnosis to make radiologic examination an essential step both in establishing the presence of an ulcer and following its course.

Peptic ulcers in infancy usually occur as acute processes, with little or no surrounding inflammation. In the newborn, hemorrhage or pneumoperitoneum may be the first indication. In older children, duodenal ulcers also present somewhat atypical clinical features. The patients complain of generalized or periumbilical abdominal pain, gastric upsets, nausea, vomiting, and little relief from food or medication. In some, radiologic examination discloses only minor mucosal changes, so that the differentiation of duodenitis from a superficial ulcer cannot be made with certainty. As the ulcers become deeper, usually in older children, the adult type of symptoms appear. Radiologic investigation of these patients will frequently disclose an adult pattern of ulcer deformity. Pyloric obstruction due to scarred duodenal ulcer is relatively uncommon in children.

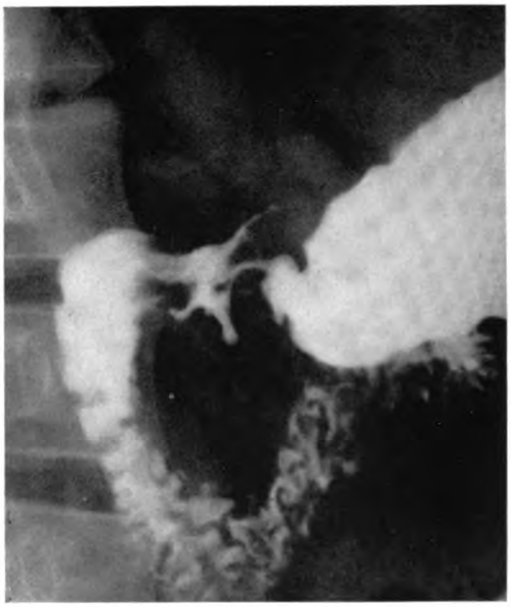

FIG. 127.—Duodenal ulcer in a 41-year-old man with constant pain in the midback area due to penetration into the pancreas. Verified at operation

A well-formed duodenal ulcer is readily diagnosed in adults. However, in the early stages, before the mucosa has been disrupted, nothing more than irritability of the bulb and granularity of the mucosa is apparent. The question as to whether duodenitis is an early stage of duodenal ulcer is not answered with assurance, but the association appears more than coincidental. The technic of examination need not be discussed in detail here. Suffice it to say that it is not sufficient for the examiner to say that he saw a deformed bulb fluoroscopically, but he must record his observations on adequate spot roentgenograms and support his visual impressions by a series of carefully planned films. No single routine is superior to another. In the search for an ulcer, due consideration must be given to the fact that while most of them occur on the lesser curvature side of the bulb, others on the posterior or anterior wall, or the greater curvature, or the ring itself, or at the apex of the bulb, or in the postbulbar area all must be considered and be looked for. The classic sign is the niche, which corresponds to the crater. However, the pathologic process usually is more

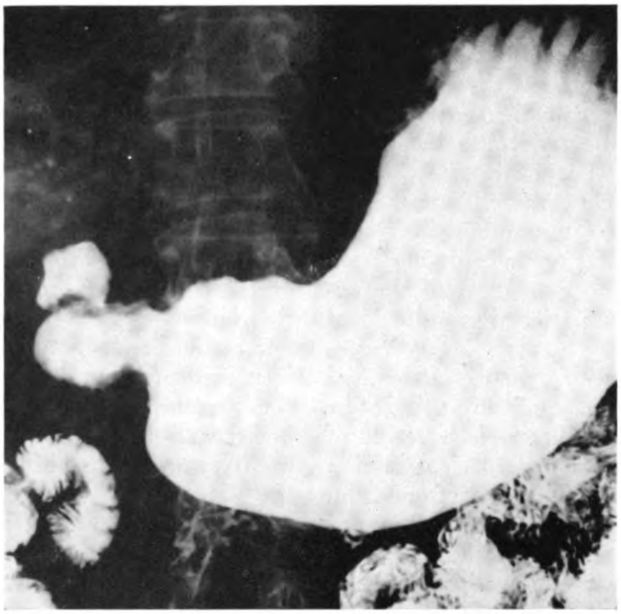

FIG. 128.—Pyloric deformity in a 66-year-old man due to extrinsic adhesions from a duodenal ulcer, verified at necropsy.

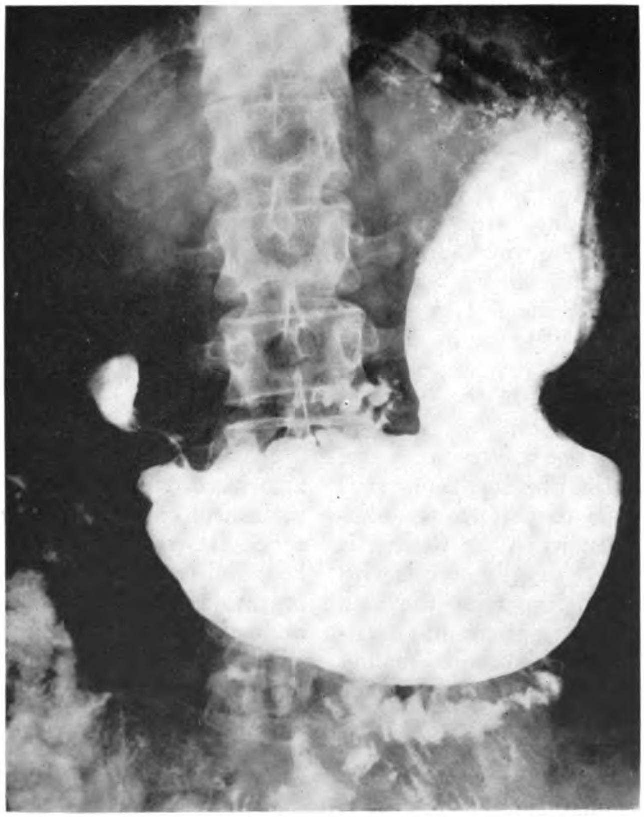

FIG. 129.—Stenosing duodenal ulcer with marked 5-hour gastric retention.

complex than a simple hole in a normal duodenum. Ulcers may be serpiginous or multiple, and their presence affects the motor activity of the stomach and the duodenum. The effects of an ulcer on the serosa and on the tissues about the pyloroduodenal area also are complex. The radiologic picture, too, is not entirely constant. The same patient minutes apart often shows ulcer configurations which differ remarkably one from another, and still the sum total of the examination is that the patient has a duodenal ulcer.

Duodenal ulcers are frequently associated with gastric retention. In many this is due to spasm, while in others it is a result of mechanical obstruction due to cicatrization of the ulcer. The differentiation of one from another requires reexamination after appropriate medical treatment in many patients. In patients who have advanced dilatation of the stomach, the condition may be apparent on plain film roentgenograms by a density which occupies the left upper and mid-abdomen extending downwards and to the midline. If sufficient fluid accumulates a mass lesion is simulated. Examination in the erect position reveals a fluid level. If this does not form the examination may well be continued after some water-soluble contrast medium is swallowed. The passage of a nasogastric tube sometimes makes the diagnosis immediately apparent as well. Cicatrizing duodenal ulcers with gastric dilatation may be is accompanied by shallow gastric ulcers along the distal part of the lesser curvature. These are difficult to identify radiologically, and usually are disclosed either on inspection of the removed specimen or at postmortem.

Duodenal ulcer is one of the common causes of gastrointestinal hemorrhage. It is not unduly hazardous to perform a radiologic examination even when the patient is bleeding vigorously. We usually do this with the patient recumbent, inspecting the area fluoroscopically after the patient has swallowed about 2 ounces of barium mixture, and terminate the examination as soon as the diagnosis is established. In some cases fluoroscopy is not possible, and then films are exposed with the patient in the right anterior oblique prone position. If the stomach is filled with clotted blood a rather bizarre appearance results. Recently, especially when penetration is suspected, we have substituted water-soluble nonabsorbable contrast media for barium.

Sudden perforation of a duodenal ulcer is the most frequent cause of spontaneous pneumoperitoneum, with severe pain and shock the outstanding symptoms. Erect roentgenograms disclose free air under one or both leaves of the diaphragm. Lateral decubitus views are useful if erect films cannot be obtained. Sometimes free air can be identified on recumbent roentgenograms by the presence of gas on either side of the bowel wall, between the kidney and spleen, the liver and right kidney or the spleen and the diaphragm. No constant relationship exists between the size of the perforation and the extent of pneumoperitoneum. Free air is less frequent with posterior wall perforations than with those of the lesser curvature or the anterior wall of the duodenal bulb. This is to be expected, because adherence of posterior perforations to adjacent viscera serves to wall off the lesion. Free intraperitoneal air can be absorbed within 5 or 6 hours, so that if a perforation becomes sealed off no air may be demonstrable radiologically after that time. Relatively small quantities of air can be demonstrated radiologically, pneumoperitoneum being visible with quantities as small as 10 to 20 cc.

Infrequently, a perforated duodenal ulcer can be identified on plain film roentgenograms. Frimann-Dahl stated that in a review of 268 cases of perforated gastric and duodenal ulcers, a niche was visible in 8 per cent without contrast examinations because of gas in the craters.

Spontaneous pneumoperitoneum often is associated with effusion of fluid into the peritoneal cavity, with ileus as part of the shock-like state which transpires. Lesser bowel distention under such conditions can imitate an obstructive pattern. If intra-abdominal fluid is present, fluid levels appear. While most ulcer perforations lead into the greater peritoneal cavity, cases are reported in which fluid accumulates in the lesser peritoneal sac. This produces a cyst-like collection, sometimes with subhepatic or periduodenal air pockets which are best demon-

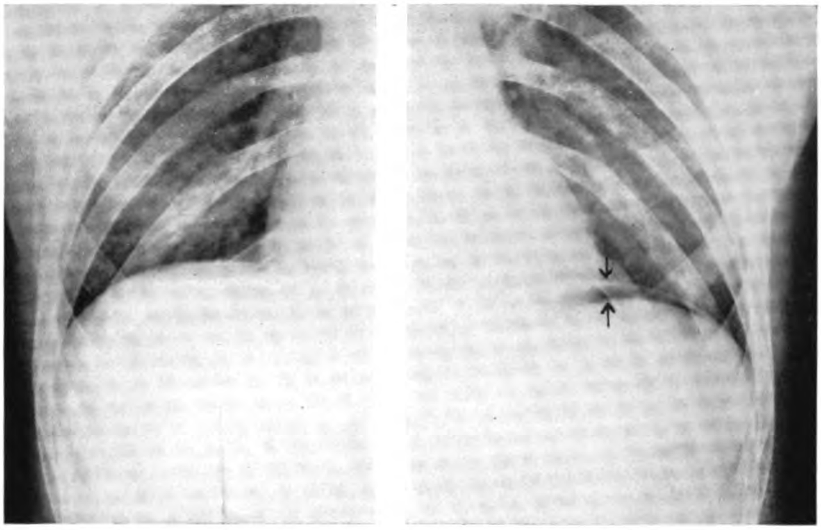

FIG. 130.—Spontaneous pneumoperitoneum due to penetrating ulcer in a 44-year-old man. More gas is present under the left than under the right diaphragm (arrows).

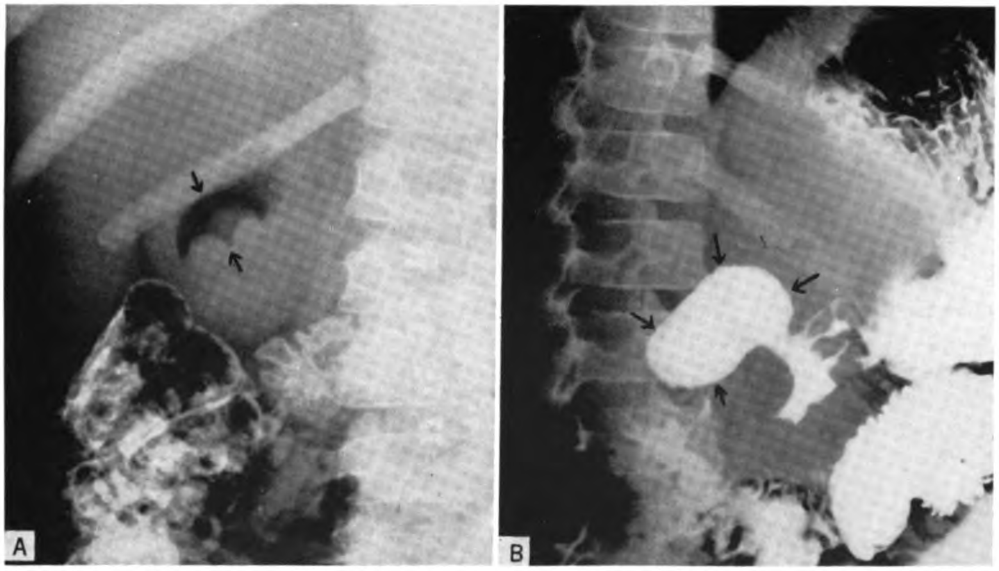

FIG. 131.—*A*, Cone-down study of right upper quadrant in a 9-year-old boy with abdominal pain and weight loss. A collection of gas is present beneath the twelfth rib (arrows).

B, Same patient, upper gastrointestinal examination shows barium in this cavity. At operation a large craterous duodenal ulcer was found. One wall was formed by the gallbladder (arrows).

strated on spot compression films. Feldman pointed out that if this is suspected, the examination should be performed before gas is absorbed.

Occasionally a peptic ulcer perforates during examination, and barium spreads through the peritoneal cavity. The patient should receive surgical attention as quickly as possible. It has been shown that barium alone is not the offending medium, but that gastric or bowel contents with infection produce the inflammatory changes in which barium becomes encysted. Hayden believed that a reflex pylorospasm takes place at perforation, which limits the passage of barium into the abdomen.

Patients with perforated peptic ulcers are regarded as acute surgical emergency cases. Nevertheless, there are instances recorded in which spontaneous cure has taken place. We have observed 3 such patients. Bertram reported 16 patients like these with no mortality. All had duodenal ulcers, and 13 had spontaneous pneumoperitoneum. One developed a subphrenic abscess which later required surgical relief.

Perforations of duodenal ulcers occasionally produce choledocho-duodenal fistulas, with gas entering the biliary tract through this opening. The clinical picture in these patients often suggest gallbladder disease. Sullivan and Hamman remarked that in some of these patients the acute symptoms were immediately and dramatically relieved by the appearance of the fistula. Choledochoduodenal fistulas due to gallbladder disease are infrequent, the usual channel in these patients being between the gallblader and the duodenum. Radiographic examination of patients with duodeno-biliary fistulas disclose the presence of gas in the biliary tract. This often is visible on plain film examinations. The entrance of opaque medium into the biliary tree is not constant. In evaluating this condition, inquiry should be made as to previous operation, especially sphincterotomy, which may permit a free passage into the biliary tree.

Occasionally duodenocolic fistulas are produced by perforated duodenal ulcers. In a report of 14 such cases, Clayton and Thornton found that the patients had complaints from a few weeks to more than 20 years, with weight loss, diarrhea and pain the outstanding symptoms. In only 4 of these patients did undigested food pass into the stool. The identification of duodenocolic fistula is better with barium enema than by gastrointestinal examination. Two cases of nonmalignant duodenocolic fistula between the third portion of the duodenum and the ascending colon were reported by Ogilvie. In each the fistula appeared to have originated in a caseating tuberculous lymph node. He mentioned that most such fistulas were secondary to carcinomas of the transverse colon, and that only 5 benign duodenocolic fistulas had been reported in the past 65 years. These were caused by duodenal ulcers in 3 patients, typhoid ulceration in one and ulcerative colitis in another.

A rare instance of a fistulous tract between the duodenum and the right kidney was reported by Stock in a 22-year-old male who complained of pain in the right loin, dysuria and frequency for about 2 years. Retrograde pyelography revealed a tract between the renal pelvis and the duodenal bulb. At operation a deep penetrating duodenal ulcer was found leading to the renal pelvis.

The most common direction of perforation of duodenal ulcer is towards the head of the pancreas. This originates as a burrowing into the duodenal wall and fixation of the serosa against the pancreas. Later, actual excavation and invasion follows, frequently associated with chronic pancreatitis. Sometimes this lesion is identified by a localized pocket of air on plain roentgenograms. More often the lesion is disclosed by persistence of barium in a markedly deformed bulb, which simulates a diverticulum because of extension beyond normal confines. When pancreatitis is present, a gentle straightening may be noted in the uppermost portion of the inner aspect of the duodenal loop with flattening of the mucosal folds in this area. Periduodenal adhesions also extend to the second portion of the duodenum. In 2 of our cases perforations at the apex of the bulb closed off the duodenum sufficiently to produce obstruction above the ampullary area. Penetrating duodenal ulcers extending to the gallbladder give rise to pericholecystoduodenal adhesions. Great difficulty sometimes is encountered in deciding whether a deformed bulb is due to adhesions from an ulcer or from those due to gallbladder disease. Combined oral cholecystography and upper gastrointestinal examinations are helpful in some of these cases.

Duodenal ulcers infrequently become as large as giant gastric ulcers, and such craters are mistaken for normal duodenal bulbs. In a review of the literature, Haml found 12 cases with craters varying from 2 to almost 6 cm. in diameter, and reported 1 with a crater 5 cm. wide. Of the 13 cases mentioned,

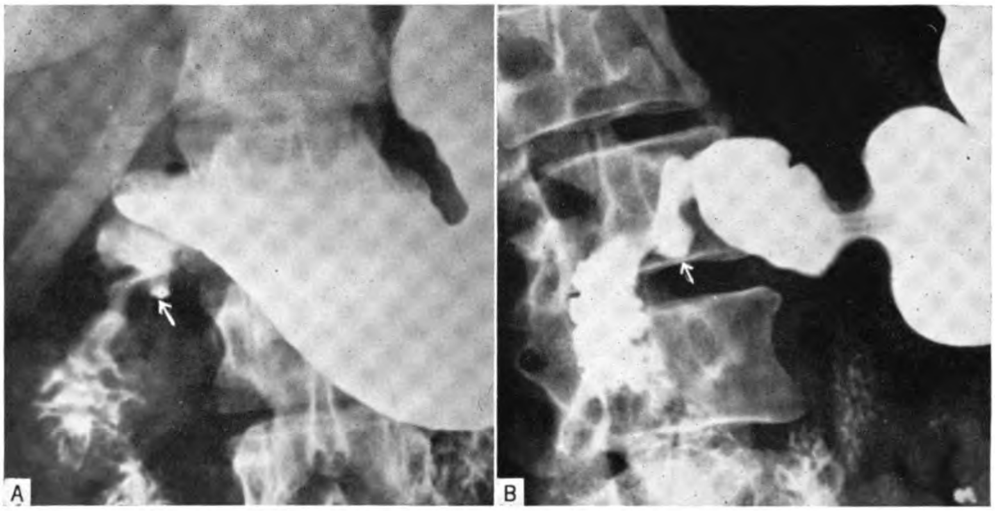

FIG. 132.—*A*, Postbulbar ulcer in a 46-year-old woman (arrow).
B, Same patient, 6 years later, after multiple attacks of abdominal pain radiating to the back. The ulcer crater is again visible, and is larger (arrow).

only 5 had been diagnosed during life. No distinctive clinical picture is associated with giant duodenal ulcer. Radiologically, the diagnosis is suggested by rapid entrance of barium into the crater, and a tendency to persist at this site in a somewhat irregular fashion. A giant duodenal ulcer was seen by us in a 10-year-old boy, whose chief complaints were abdominal pain and weight loss. The bulb was irregular, and a large cavity protruded from the superior aspect of the scarred bulb which at first glance was believed to be an atypical diverticulum. This was 6 cm. long and 3 cm. wide. At operation a large craterous ulcer was found, one wall being formed by the gallbladder. Gas had collected in the ulcer crater as seen on scout films, but the nature of the lesion was not clearly recognized until a barium meal had been given.

The term postbulbar duodenal ulcer refers to those involving the second portion of the duodenum from the apex of the bulb to the region of the ampulla of Vater. They diminish in frequency caudally, occur mainly on the medial posterior wall of the upper portion of the descending duodenum, and constitute from 5 to 20 per cent of duodenal ulcers as reported in various series. According to Swarts and Rice, the incidence of postbulbar ulcers at necropsy is approximately 9.5 per cent. As a rule the clinical history is atypical in that pain is apt to be more severe, but periodicity of symptoms is lacking. A resemblance to the symptoms of gallbladder disease has been noted. Postbulbar ulcers are frequently associated with hemorrhage, and their response to medical treatment is not as satisfactory as with bulb ulcers. They often penetrate towards the head of the pancreas, with a boring pain which is unrelieved by food or medication. Obstruction due to postbulbar ulcers is not as frequent as with cicatrizing lesions in the pyloroduodenal area.

Radiologic recognition of postbulbar ulcers requires careful compression studies of the apex of the bulb. Because of the tendency of the duodenal loop to curve back upon itself, oblique and lateral views are essential. The ulcer may be overlooked unless films are obtained with the duodenal loop partly filled. With perforation, barium may persist in the niche, and periduodenal fibrosis may be followed by narrowing and fixation of the descending duodenum. Irritability and hypermotility of the pyloroduodenal segment is frequent. Spontaneous pneumoperitoneum due to perforation of a postbulbar ulcer is uncommon.

The coexistence of duodenal ulcers and gastric cancer is relatively infrequent. Fischer, Clagett and McDonald noted that 48 such cases were seen between 1911 and 1945 at the Mayo Clinic, but during that time there were 45,000 cases of duodenal ulcer and 13,000 gastric carcinomas. A correct diagnosis was made in 22 of the 46 cases studied. The gastric lesion was missed in 5, and the duodenal ulcer overlooked in 11 others.

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MARGINAL ULCERS

Stomal ulcers were more common when the operation of choice for duodenal ulcer was gastroenterostomy. These appeared at varying times. About one-third occurred within a year, another third within two years, and the others up to 15 years or even longer after operation. Since the advent of subtotal gastrectomy, gastrojejunostomy and vagotomy as the favored operations, the incidence of marginal ulcer as a postoperative complication has diminished. The symptoms often are similar to those present before operation. Pain may be quite severe, and bleeding or perforation is frequent. Pneumoperitoneum is uncommon, most of the perforations being confined by the adjacent soft tissue.

Marginal ulcers form at the site of anastomosis, either on the gastric or jejunal side. Those in the afferent jejunal loop are more frequent than in the efferent loop, presumably because of the high acid content in the former. The same general pattern in the development of a peptic ulcer occurs, with early mucosal inflammation, crater formation and later penetration and perforation. Fistulous tracts to the transverse colon are relatively late occurrences. The colonic contents enter the stomach more readily than the gastric contents pass into the colon because of a valve-like flap at the stoma. For this reason barium enema examination is more productive in the identification of gastrojejunocolic fistula than an upper gastrointestinal investigation.

The radiologic diagnosis of stomal ulcer often is vexing. A fleck of barium identified in a defect either at the stomal line or the afferent loop is diagnostic. The identification of an ulcer niche in the stomach or efferent loop is more difficult. In this examination inspection of the mucosal pattern after the first swallow of barium is most important, and serial spot roentgenograms in several angles of projection are required. With a well-functioning stoma, the contrast material flows rapidly into the jejunum, mainly into the efferent loop and thence into the distal jejunum. The amount of barium entering the afferent loop is quite variable, and retrograde passage to the proximal duodenum is not infrequent

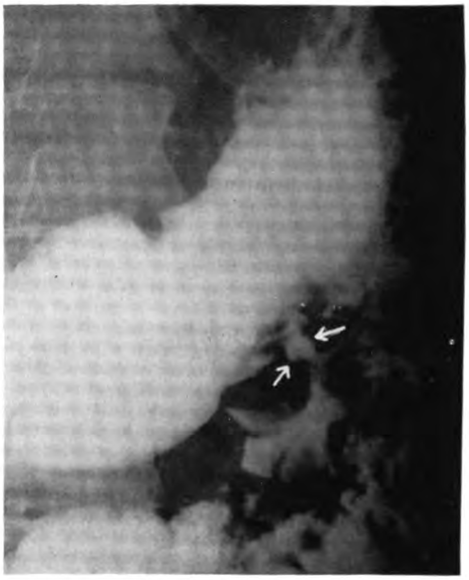

FIG. 133.—Gastrojejunal ulcer in a 58-year-old man with gastrojejunostomy. The crater was in the efferent loop of jejunum, and extended to the stomal line (arrows).

The presence of a gastrojejunocolic fistula occasionally is suggested by diarrhea, pain, vomiting, weight loss and a rapid nutritional deficiency. When the colonic contents enter the stomach a foul breath and fecal vomiting may occur. This almost always is a late complication of marginal ulcer. Other causes are perforations of colonic or gastric carcinomas, and rarely a benign greater curvature ulcer produces a similar lesion.

Infrequently gastric carcinoma appears at the gastroenterostomy site, with a clinical picture like that of a stomal ulcer. The diagnosis is suggested radiologically by rigidity of the involved gastric wall, sometimes with protrusion of tumor into the remaining gastric cavity.

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ACUTE GASTROENTERITIS

The clinical picture of acute gastroenteritis is so characteristic that radiographic examinations usually are not required. Plain films of the abdomen in such patients disclose nonspecific distention of the lesser and large bowel. Barium studies reveal flocculation of barium, hypermotility, widening and irregularity of the valves of Kirkring, and occasional areas of dilatation and smoothening of the lesser bowel. Occasionally the picture simulates lesser bowel obstruction, with progressive intestinal dilatation and fluid level formations which persist for a few days. The situation was so questionable in a few of our patients that operation was deemed necessary. These patients had not responded to intubation, and progressive pain and vomiting became alarming. At operation, no obstruction was found. Only a small amount of free fluid was present in the peritoneal cavity, and the serosa was glistening. The complaints subsided during the next few days. Gastrointestinal examination about 10 days thereafter failed to reveal any specific changes. Since then we have found that examination with water-soluble, nonabsorbable contrast media useful in determining whether obstruction is present in sufficient degree to warrant operation. The interpretation of these examinations requires understanding of the processes of partial obstruction. They make it possible to delineate clearly which loops of intestine are distended, and passage of the contrast material into the colon within 1 to 2 hours is good evidence against a significant obstruction.

Necrosing enteritis is an unusual form of acute lesser bowel inflammation which occurs suddenly, simulating perforated ulcer, acute appendicitis or some other abdominal emergency. The correct diagnosis is seldom made clinically. The patient sometimes expires within 24 hours, but if they survive blood-stained diarrhea with paralytic ileus may appear. Most of these lesions occur in the jejunum, with perforations and peritonitis as further complications. Brynjulfsen terms the severe form of this condition jejunitis acuta phlegmonosa, and the milder form jejunitis acuta simplex. The disease is believed to be secondary to mucosal invasion by virulent microorganisms, with later involvement of the entire bowel wall resulting in marked infiltration and edema. Small hemorrhages and fibrin deposits occur in the mesentery, and lymphangitis and lymphadenitis are found commonly. The pathologic changes diminish distally, with interspersed skip area of normal intestine. Prevot observed that the condition is more frequent in Europe, particularly in adults who had been subjected to malnutrition.

An acute form of regional enteritis in children was described by Storrs and Hoekelmann. They encountered 28 such cases in children up to 15 years old, and added 8 more. In their opinion this condition was different from the acute enteritis observed in adults.

The radiologic picture of necrotizing enteritis reflects the acute inflammation of the bowel wall. Plain film roentgenograms reveal irregularly distended, fixed loops of jejunum in the left upper quadrant. The mucosa may be jagged in appearance, and fluid levels appear on erect studies. Barium examinations disclose obliteration of the valves of Kerkring, and irregularity of the jejunal walls is more apparent than on plain films. When ileus is present the distal bowel also is dilated.

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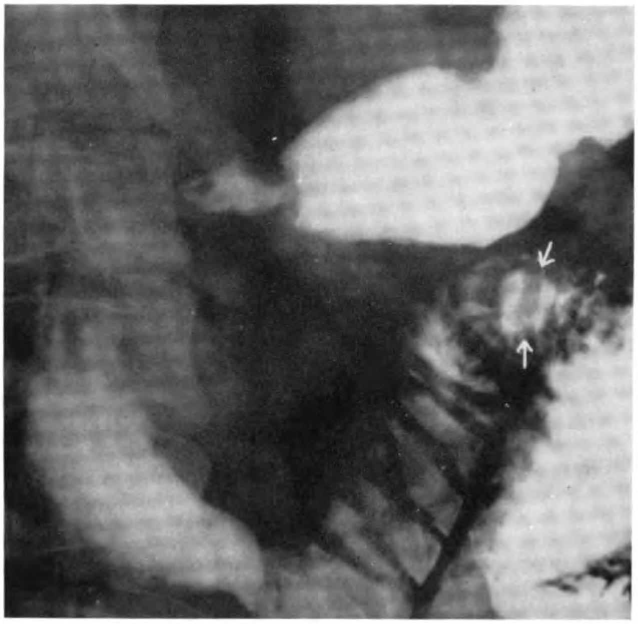

FIG. 134.—Jejunal ulcer (arrows) due to ulcerogenic tumor of the pancreas, verified at necropsy. This 39-year-old man had several postoperative recurrences, and expired after a recurrent ulcer perforated suddenly.

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ULCERS OF THE SMALL INTESTINE

Benign ulceration of ectopic gastric mucosa occurs in areas where these rests are frequent, especially in Meckel's diverticula. The association of peptic ulcer with ulcerogenic tumors of the pancreas has been mentioned before. In a recent report Ellison collected 24 such cases from the literature. Of 30 ulcers, 4 were primary in the jejunum. This syndrome is characterized by excessive acid secretion and rapidly progressive atypically located ulcers which recur after surgery and often are resistant to the most heroic treatment. We saw one such patient, a 39-year-old robust male, who had had a 4-year history of ulcer pain which responded to medical treatment. He was admitted to the hospital because of severe midback pain which had become intractable. On radiologic examination an ulcer was found at the duodenojejunal junction. Apparently perforation had occurred, inasmuch as the lesion was fixed in position. This was confirmed at operation. Subsequently he developed another large ulcer at the site of resection requiring another operation. The ulcers again recurred, and he finally expired after perforation and hemorrhage from one of the recurrent ulcers in the jejunum. At necropsy 2 non-insulin producing islet cell adenomas were found in the head of the pancreas.

More common than these are nonspecific ulcers of the jejunum and ileum, seen most often in the ileum as solitary lesions varying in size from about 0.3 to 4 cm. in diameter with linear, stellate or oval craters. No known etiology is held responsible, investigations into the role of peptic and tryptic digestion, infection, infarction and trauma all being inconclusive. The clinical picture

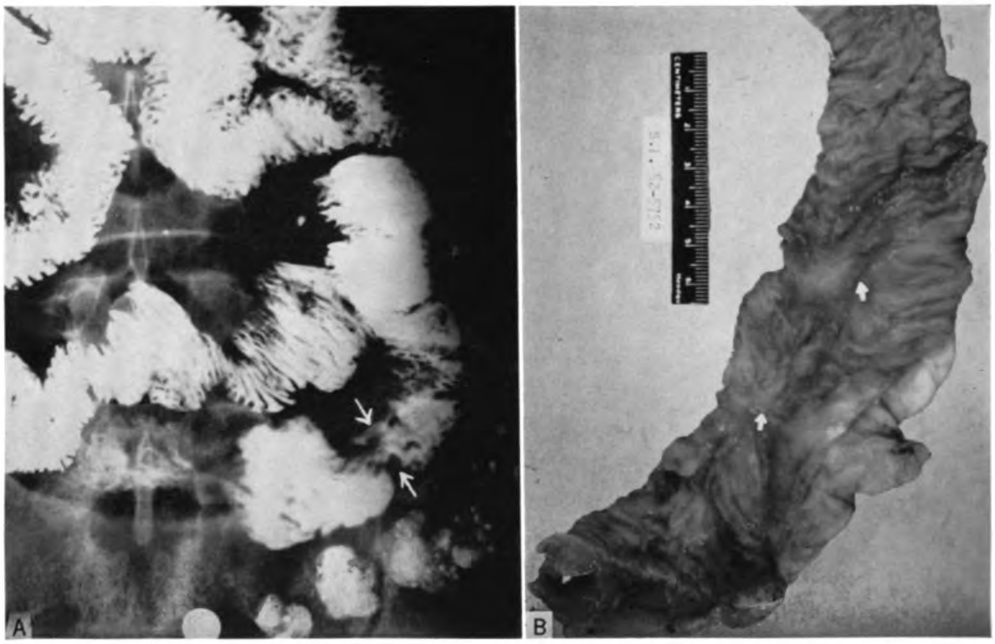

FIG. 135.—*A*, Jejunal ulcer in a 39-year-old man with abdominal pain radiating to the back, and persistent anemia. Lesser bowel examination discloses a fixed irregular area just above the left sacroiliac joint (arrows). *B*, Operative specimen. The knuckling of bowel was due to serosal adhesions. The arrows indicate two small ulcer craters. (Muehlbauer, M., Kramer, A., and Epstein, B., courtesy of Am. J. Digest. Dis., *21*, 77, 1954).

reflects the possibilities of hemorrhage, obstruction or perforation. Morlock, Goehrs and Dockerty reviewed 18 cases, with a a follow-up of 14 previously reported. The most common complaints were related to intestinal obstruction. Bleeding was not as prominent as had been mentioned by others. A case in which slow bleeding resulted in profound anemia and weakness was reported by Muehlbauer, Kramer and Epstein. This patient had been examined repeatedly, and the diagnosis of a psychosomatic disorder was constantly confounded by the undeniable anemia. The correct diagnosis finally was established by identifying a kinked, relatively fixed loop of bowel in the left lower abdomen. At operation this loop was fixed by adhesions from a penetrating ileal ulcer to the adjacent soft tissues. The patient's recovery after resection of the lesion was remarkable. As a rule, the radiologic demonstration of jejunal and ileal ulcers is difficult. The diagnosis is established at surgery, usually performed for intestinal obstruction or for bleeding.

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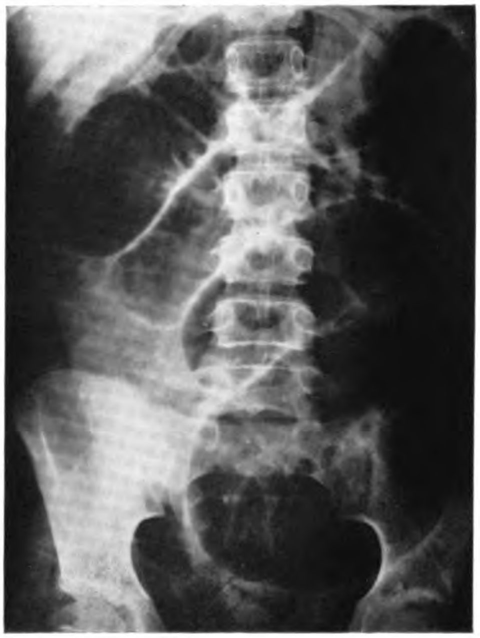

FIG. 136.—Postoperative ileus in an 11-year-old child 1 week after appendectomy. Recovery with conservative treatment.

APPENDICITIS

Acute appendicitis often presents bizarre symptoms, particularly in very young and elderly patients. As a rule, the diagnosis is established clinically, and scout films of the abdomen early in the disease disclose nothing of importance. With serosal inflammation and exudation of fluid, distention of the lesser bowel in a widely varying pattern of early ileus appears. Later, ileus becomes more pronounced, and the degree of dilatation sometimes cannot be distinguished from that associated with intestinal obstruction. A sentinel loop of dilated small intestine occasionally appears near the acutely inflamed appendix. This is not an absolute sign, and in our experience has been unreliable. Occasionally a single distended loop is separated appreciably from the inflamed appendix, and probably represents a phase of ileus. Both the sentinel loop and those present with early ileus are transitory.

With perforation and peritonitis ileus often becomes severe. Spontaneous pneumoperitoneum has been observed with perforation in isolated cases. Three such patients were seen by us. In a review of 32 cases of pneumoperitoneum due to conditions other than peptic ulcer Spensley, Nelson and Childs found only 1 due to a perforated appendix. As with peritonitis of other origin, the properitoneal fat line becomes hazy or disappears, but this is inconstant. The right psoas shadow likewise may become less sharp, and the lumbar spine occasionally is convex towards the left.

As a rule barium enema examination is not done when acute appendicitis is suspected. However, in some cases with doubtful diagnosis, such examinations have disclosed irritability of the cecum and ascending colon, and absence of filling of the appendix.

The question of identifying the effects of previous appendiceal inflammations radiologically often arises. It has been reported, and we too have observed, that the appendix under such circumstances may fill poorly and be relatively fixed in position. However, the specific significance of these changes cannot be regarded as entirely reliable.

Acute appendicitis which has run the full course from blockage of the lumen to perforation often terminates in an appendiceal abscess. Sometimes this is identified on scout films as a soft tissue mass impressed against the gas-distended cecum. If the abscess is walled off, and the acute inflammation subsides, further examination by means of barium enema discloses a relatively smooth cecal filling defect secondary to the adjacent mass. Occasionally a rather irregular protrusion is produced, simulating neoplasm. Considerable irritability of the cecum and ascending colon is noted fluoroscopically. As a rule, the appendix itself does not fill. Additional investigation by means of gastrointestinal examination serves to locate the terminal ileum in relationship to the mass, and also serves to exclude intrinsic disease such as regional enteritis or tuberculosis.

In some cases perforation of the appendix retrocecally produces retroperitoneal abscess. In such patients scout films disclose a collection of gas adjacent to the cecum, and interstitial emphysema extends to the psoas muscles and to the kidney. In a review of 65 cases of lumbar and iliac retroperitoneal

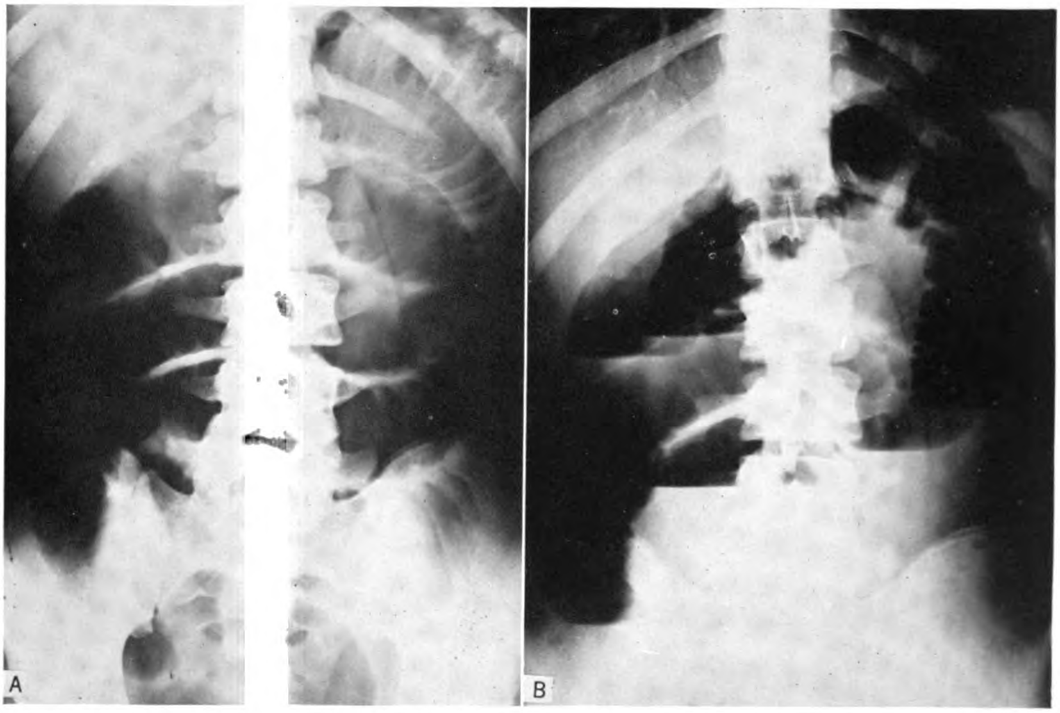

FIG. 137.—*A*, Ileal obstruction due to adhesions in right lower quadrant 1 year after appendectomy. Supine film reveals diffusely dilated lesser bowel, with no gas in the colon.

B, Same patient, erect film showing numerous fluid levels.

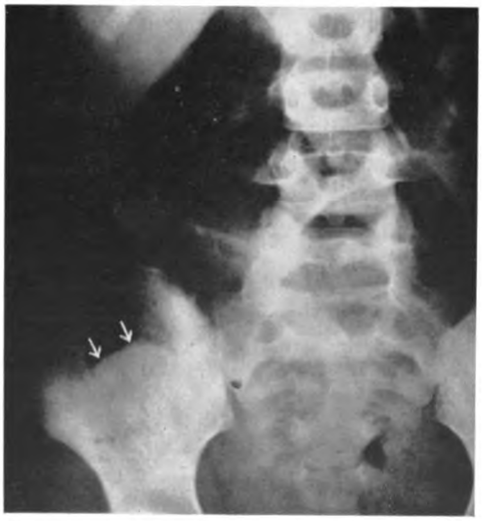

FIG. 138.—Appendiceal abscess in a 5-year-old boy with abdominal pain, diarrhea and intermittent cramps. Note the defect in the gas-filled cecum (arrows). Verified at operation.

abscess and diffuse phlegmon, Neuhof and Arnheim noted that 12 had been caused by retroperitoneal rupture of the appendix.

Cecal gangrene with acute appendicitis occasionally involves the terminal ileum, rendering this structure relatively rigid. The formation of adhesions in this area may be followed by rapidly progressive intestinal obstruction. Another rare complication is fistula formation. More often a fistula in the right lower quadrant is a complication of appendectomy undertaken mistakenly for regional or tuberculous enteritis, neoplastic or granulomatous cecal disease. An instance of post-appendicitis fistula was reported by Hurwitt and Lentino, who remarked that they were unable to find a similar case in the literature.

We had a 54-year-old man who had a perforated gangrenous appendix in whom a sinus tract appeared 4 months after appendectomy with drainage of an abscess.

Instillation of lipiodol showed communication with the sigmoid, but the patient did not drain fecal material. At operation, the cecum was surrounded by a large granulomatous mass.

Obstructive phenomena associated with acute appendicitis are usually due to ileus. Following operation, an obstructive picture again may be due to ileus. This diagnosis may be difficult, and mechanical obstruction due to adhesions can supervene relatively quickly. Sometimes this block is overcome readily with small intestinal drainage through a decompressive tube. More often, patients develop intestinal obstruction months or years after operation. The plain roentgenograms disclose a pattern of lesser bowel distention with gas and fluid levels. These patients also respond to conservative treatment, but great caution should be exercised in delaying too long before surgical relief is offered.

Calcium-containing fecaliths as well as those without calcium are quite common in the appendix. These can be significant in the development of acute appendicitis by obstructing its lumen. Berg and Berg reported that they saw 42 such cases in 5 years. The presence of a coprolith in a patient with acute abdominal symptoms is believed to offer a 90 per cent chance of acute appen-

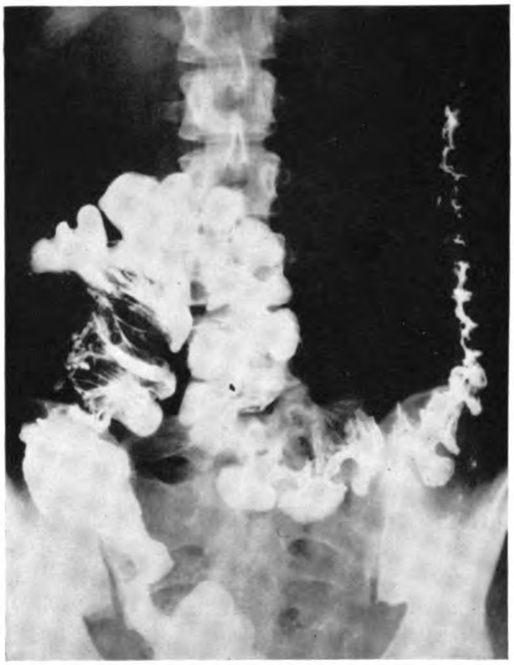

FIG. 139.—Retrocolic abscess in right side of abdomen 3 months after appendectomy in a 32-year-old woman. A mass was felt in the right side just above the iliac crest. Verified at operation.

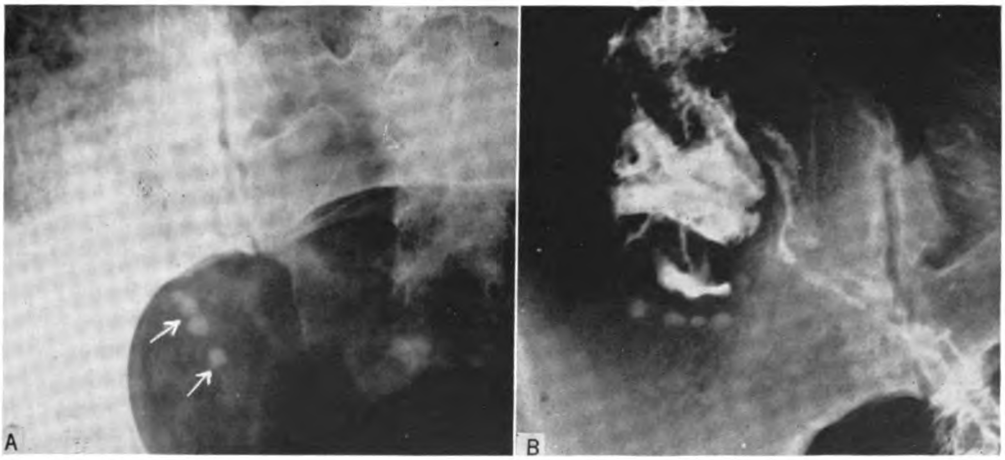

FIG. 140.—*A*, A row of four appendiceal stones (arrows) in a 46-year-old man. No symptoms were attributed to these.

B, Same patient, 5 days later. The position of the stones has changed. Appendectomy done when patient was operated upon for gallstones. No inflammation was found in the appendix.

dicitis, and in about half the appendix is either gangrenous or perforated. Acute symptoms with coproliths are somewhat more frequent in younger patients. These stones are formed by inspissated fecal matter, and calcification does not appear unless calcium salts are deposited as a result of inflammation of surrounding tissue. Occasionally, coproliths are laminated because of intermittent deposition of calcium.

Appendiceal stones usually are small, but reach diameters of over 2 cm. Small coproliths are easily confused with ureteral stones or phleboliths, and occasionally assume configurations indistinguishable from small lymph node calcifications. Multiple fecaliths are not uncommon. In one of our cases multiple facetted appendiceal stones were present, the largest measuring 2.3 cm. in length and about 1 cm wide. The positions of the stones within the abdomen vary with the situation of the appendix. Not infrequently, barium will be retained for several weeks after gastrointestinal examination. Most of the time this eventually disappears without ill consequence.

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PSEUDOMEMBRANOUS ENTEROCOLITIS

The association of severe enterocolitis with antibiotic therapy has been stressed recently. There is little question that this condition has become more frequent with the use of potent antibiotics, but it was known as a serious disease before the advent of these drugs. A poor nutritional condition particularly in patients operated upon for gastrointestinal neoplasms is believed contributory. However, the disease occurs in patients who have not been operated upon as well as those who have had antibiotic treatment and operation.

Pseudomembranous enterocolitis occurs in both mild and severe forms. In the latter particularly the prognosis is bad. The condition usually appears within a week after operation, and is characterized by violent diarrhea, abdominal pain and distention, shock and dehydration. Death may occur rapidly. The disease also occurs without diarrhea, but at necropsy the intestines of these patients are found full of fluid. Enterocolitis is easily confused with other postoperative complications, particularly peritonitis and intestinal obstruction. It is believed that the antibiotics which predispose towards this disease so alter the intestinal bacterial flora as to permit the development

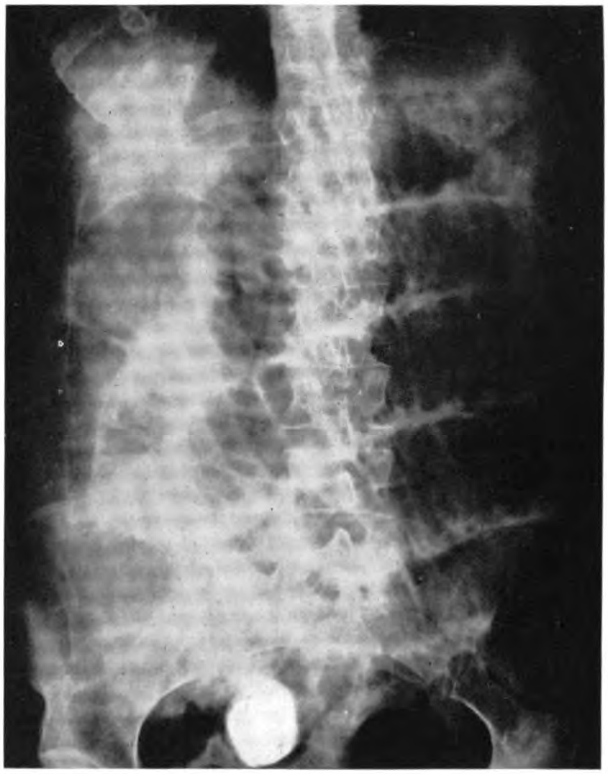

FIG. 141.—Ileus due to acute necrotizing enterocolitis following hemi-colectomy for carcinoma of the hepatic flexure. The diffuse ileus suggested the presence of intestinal obstruction. Diagnosis verified at postmortem.

of a micrococcus organism which is highly resistant. Other organisms also have been identified.

The pathologic changes often are extensive, involving the stomach and duodenum as well as the bowel in some cases. Essentially, the lesion is a necrotizing purulent ulceration of the mucosa, producing either plaques or extensive pseudomembranous elevation of yellow-brown fibrinous edematous mucosa. Occasionally submucosal hemorrhage and considerable intestinal bleeding have been encountered. There are no specific radiologic changes. The diffuse ileus is readily confused with gaseous and fluid distention like that seen with intestinal obstruction in some cases. In the early phases of the disease the pattern of lesser bowel distention may be less pronounced. In some cases, particularly those without diarrhea, the fluid filled distended loops of lesser bowel are prominent.

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NONSPECIFIC CECAL AND COLONIC ULCERS

A patient believed to have acute appendicitis occasionally is found at operation to have an acute cecal ulcer. These appear as discrete, sharply punched out lesions, or a diffuse phlegmonous process with acute and chronic inflammation of the bowel may be present. With perforation either localized or diffuse peritonitis develops. The radiologic diagnosis of cecal ulcer cannot be made with certainty, the signs being those of an acute intra-abdominal inflammation. With pericolic and pericecal extension, a mass simulating neoplasm often is present. Ten cases of acute nonspecific cecal inflammation were reported by DeCamp and Penick. In 5, ulcers were present, and another was presumed to have an ulcer when a fecal fistula developed after simple drainage. Perforation occurred in 3 cases, with a localized abscess in one and widespread sepsis and death in the other 2. The importance of antibiotic therapy was stressed.

Simple ulcers similar to peptic ulcers, with clean-cut edges, necrotic bases and normal surrounding mucosa, are found in the transverse colon and sigmoid as well as in the cecum. These lesions may clear spontaneously, or go on to perforation. In occasional cases, a stenotic process follows leakage, producing an appearance suggestive of a neoplasm.

Simple ulcers of the colon should be differentiated from stercoral ulcers due to constant pressure of impacted stool within the bowel, nor should they be mistaken for amebic, tuberculous or syphilitic ulcers. The identification of organisms in the stool readily serves to identify amebic ulcers. Tuberculosis and syphilis produce more extensive lesions, as a rule associated with small intestinal changes.

In our material there were 3 cases of simple colonic ulcers, 2 involving the cecum and the other the transverse colon. The preoperative diagnosis in 2 was colonic carcinoma. In the second patient, a 70-year-old woman, a partial obstruction was present, and at operation the proximal transverse colon was encircled by a granulomatous mass in which the ulcer was found after the specimen had been opened. This patient had had repeated bright red rectal bleeding for about 2 years. The third patient had a spontaneous pneumoperitoneum and ileal obstruction due to perforation of a punched-out cecal ulcer associated with localized pelvic peritonitis.

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MUCOUS COLITIS

Not infrequently patients whose main complaints are frequent soft stools with mucous, or expulsion of mucous with flatus, are referred for colon examination. Very rarely some bleeding is also noted. There is no known etiology for this disorder, Poppel, Adler, Jacobson, Stein and Lawrence suggesting that it might be a form of psychosomatic disease. Colon examination usually shows no organic changes. Radiolucent defects in the barium column and on postevacuation films may indicate the presence of thickened mucosa with some distortion of folds. The filling defects may be associated with mucous globs. There is no known relationship between this condition and ulcerative colitis.

Occasionally patients who have been taking cathartics for a long time show irritability and spasm of the bowel, with smoothening of its walls and incomplete evacuation as seen on post-evacuation films. The picture sometimes is suggestive of colitis. However, sigmoidoscopy fails to disclose any erosion of the mucosa. This condition is more frequent in elderly patients, in whom smoothening of the distal half of the colon is not at all infrequent. These changes should not be considered indicative of colitis, and due cognizance should be made as to the prolonged use of cathartics.

ULCERATIVE COLITIS

Ulcerative colitis is a frequent, unpredictable and widely varying disease observed from infancy well into adult life. Cullinan and McDougall, in a review of 346 cases, divided these patients into three groups in accordance with the bowel area involvement. Thirty-eight per cent had disease of the entire colon. In 42 per cent only part of the colon continuous distally to the rectum was affected. In twenty per cent sigmoidoscopic examination disclosed ulcerative colitis, but the radiologic investigations were normal. Cases of regional and segmental colitis were omitted from this series. All three were regarded as the same basic disease, and histologic examination of rectal biopsies were identical in all. Transition from one group to another was not uncommon in patients who initially had no radiologic changes and later progressed to involvement of the entire colon. These changes bore no specific relationship to the duration of the disease, and a sudden transition from a mild to a severe form was not unusual. The age of onset varies from about 4 years to old age, but most often patients between 18 and 30 years old are affected, women being somewhat more susceptible. The severity of symptoms and the appearance of remissions is unpredictable. The prognosis is worse when the entire bowel is affected, but even in these no constancy is encountered in the clinical picture.

In the group with normal radiologic but positive sigmoidoscopic changes, the onset may be relatively mild with only loose stools and some bleeding. There is no way of determining how or when the disease will progress to more extensive inflammation of the bowel, with more persistent diarrhea and increased bleeding. These patients also develop systemic manifestations, with a tendency to anemia. When the entire colon is involved, diarrhea often is severe, the patient becomes toxic and emaciated, with dehydration, high fever and ultimate exhaustion. It is not possible to correlate accurately the extent of the disease as noted radiologically with the condition of the patient.

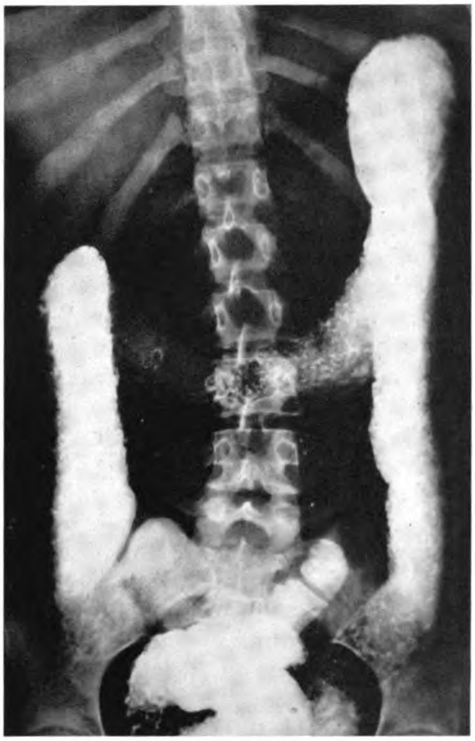

FIG. 142.—Acute ulcerative colitis in a 14-year-old girl with diarrhea for about 2 weeks. A leukemoid reaction was present in the bone marrow, and the diagnosis at first was leukemia. The colon is extensively involved with numerous ulcer craters. Pseudopolyposis is seen in the transverse descending and sigmoid colon.

The cause of ulcerative colitis is unknown. A possible psychiatric aspect to its onset and exacerbation is considered likely. The disease can occur as an acute fulminating process, but more often is chronic and intermittent. With the acute stage complications such as hemorrhage, local perforations of the bowel, fistulas about the anus and carcinomatous degeneration appear. In acutely ill patients, perforations may give only slight warning clinically. Sometimes the radiologic demonstration of relatively localized atonic bowel is an indication of impending perforation. Systemic manifestations such as arthritis and uveitis are sometimes observed. Some complications require operative relief, particularly perforation and bleeding. When the disease is intractable and uninfluenced by medical treatment, total colectomy and ileostomy may be lifesaving. The more chronic complications which require surgery are perirectal fistulas and abscesses and carcinomatous degeneration.

Ulcerative colitis is a more severe disease in children than in adults. While the usual age of onset is around 4 years, cases have been reported in much younger patients. Beranbaum and Waldron reported a 3-day-old infant with chronic ulcerative colitis. Barium enema examination at about 21 days of age disclosed multiple mucosal irregularities and ulceration, tubulation of the bowel and absent haustrations. A diagnosis of small bowel obstruction was made. At operation the terminal ileum, the cecum, the ascending colon and part of the transverse colon were thick, inflamed and friable. Necropsy disclosed ulcerative colitis and acute ileitis. Hodgson and Kennedy noted that chronic ulcerative colitis was more frequent in males, and reported on 11 cases between birth and 4 years of age. Twenty-eight others occurred between 5 and 9 years, and from 10 to 15 years of age there were 83 cases. Early adolescence was found a particularly dangerous time because of rapid progression and poor response. In general, the response to treatment in children is less satisfactory than in young adults, with a tendency towards impairment of growth and well-being.

Barium enema examination is the favored method of diagnosis radiologically. In early cases, no definite changes may be present even though sigmoidoscopy reveals definite mucosal changes. In the second group, with involvement of the distal colon, radiographic examination discloses a rapid passage of contrast medium through the involved area, with smoothening of the bowel and small indentations in the mucosa indicative of small ulcers. Post-evacuation films sometimes show no gross change in the mucosal pattern, or the folds may be thickened and straightened. More often the mucosal pattern is disrupted and irregular. With further ulceration, small pockets of barium are scattered in the involved bowel in ulcer craters.

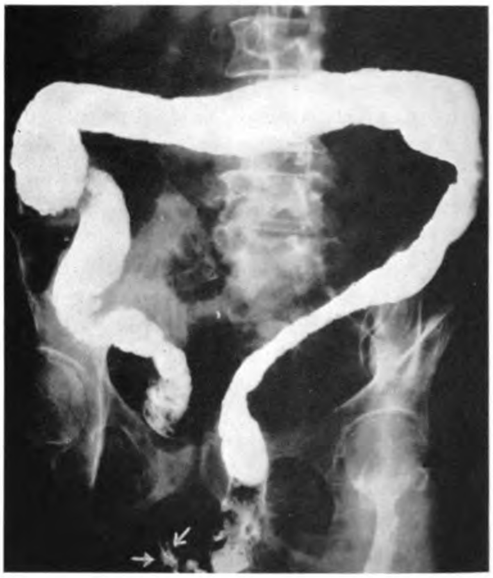

FIG. 143.—Chronic ulcerative colitis in a 58-year-old woman, known for more than 12 years. A rectoperineal fistula (arrows) extends towards the right. The colon is shortened, tubular and serrations indicative of ulcers are seen along its walls. Marked clubbing of the fingers and toes was present.

With extensive colonic ulceration, involvement extends from rectum to cecum, and the terminal ileal coil is affected in some patients. A shortening of the bowel appears, the colon becoming tubular, smoothened and ultimately quite rigid. Extensive mucosal involvement may be present before the stage of rigidity takes place. Erosion of mucosa leaves areas of relatively intact membrane, forming pseudo-polyps interspersed with shaggy, irregular mucosal lining.

Involvement of the terminal ileum occurs as extension of the inflammation to the mucosa. In a review of 107 cases of ulcerative colitis, Counsell found 18 with a complicating retrograde or "backwash" ileitis, an incidence of 17 per cent. The duration of the disease varied from a few months to over

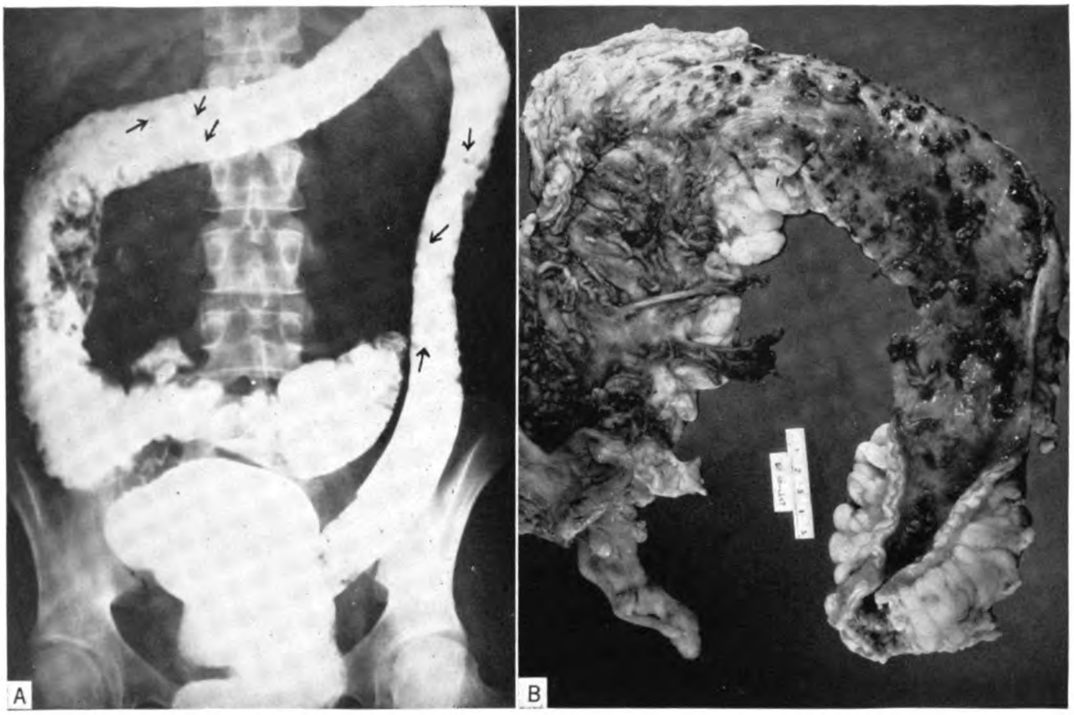

FIG. 144.—*A*, Advanced ulcerative colitis with pseudopolyposis in a 19-year-old girl with diarrhea and bloody stools for 5 years. Note the irregular markings in the ascending, transverse and descending colon. The circular negative shadows (arrows) are much more marked on the original films and indicate multiple nodular pseudopolyps.

B, Specimen of colon following colectomy. Numerous hemorrhagic pseudopolyps were present throughout the colon. Some were long and slender, but mainly were sessile. Others were free from the underlying muscularis and were like pieces of cord unattached for most of their length.

30 years, and no definite relationship was established between the severity of the ileal disease and the history of ulcerative colitis. It appeared that retrograde ileitis was more frequent with chronic continuous or slightly remittent disease without acute exacerbations. In none of the patients with ileal involvement was there more than a minor degree of pseudopolyposis. The radiologic diagnosis of ileal extension is difficult, and is usually made only in the more severe cases. The change produced is one of loss of mucosal markings with a mild ileal dilatation. The ileocecal valve may be edematous, presenting as a rather pointed channel delimited by the swollen radiolucent lips. Counsell regarded this complication as not necessarily serious. McCready, Bargen, Dockerty and Waugh discussed ileal extension of chronic ulcerative colitis on the basis of 23 postmortem examinations and 6 surgical specimens. These authors noted that about 28 per cent of 103 cases of chronic ulcerative colitis had ileal involvement. In 22 verified cases, the terminal ileum had the same gross pathologic changes as the colon. In 7 others ulcers appeared intermittently in the ileum, with normal intervening mucosa. As a rule the ileal reactions were less advanced than those in the colon. In severe cases the inflammation extended through the ileocecal valve as long longitudinal ulcerations distributed over the lumen without stenosis or thickening of the involved small bowel. On microscopic examination ulceration and denudation of the mucosa was found. In some there was perforation of the bowel wall. Barium enema examinations in 19 of the 29 cases examined histologically disclosed ileal involvement in only 4, and a

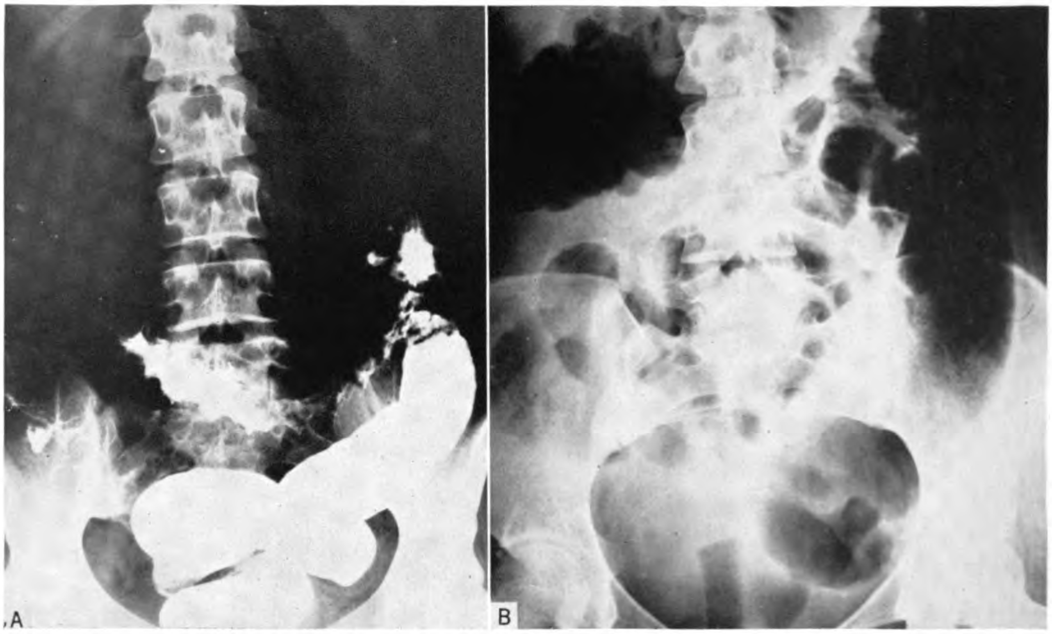

FIG. 145.—*A*, Acute and chronic ulcerative colitis in a 28-year-old woman with recurrent diarrhea and fever for 15 years. Present episode with severe bleeding.

B, Same patient 2 weeks later, scout abdominal film reveals marked distention of transverse and descending colon, with ileus. This was considered indicative of impending perforation. At operation the bowel was thin and friable and blood stained fluid was present in the peritoneal cavity although no specific points of perforation were found. Total colectomy was performed.

review of the films later showed an additional 6. The average length of involvement was about 1 foot.

The question as to whether colonic changes are reversible during periods of remission is of interest. It is possible for the colon to return to a fairly normal appearance in mild cases, and occasionally even a fairly extensive involvement may regress markedly. Such a case was reported by McEwen and Lodnell in a 23-year-old man in whom examinations 2 weeks apart showed a remarkable improvement. In a study of 46 cases followed from 2 to 16 years Ricketts, Kirsner and Palmer noted that relatively little change occurred in 30, and that in 11 the lesion was progressive. Improvement with return of haustrations and distensibility was observed in 5 cases. The radiologic reversal of colitis is not an indication of cure, inasmuch as recurrences are frequent.

Radiologic examination of the abdomen in patients with acute colitis is essential. One indication for almost immediate operation is impending perforation. This is not infrequent, and steroid treatment has been regarded as a precipitating factor. However, Goldgraber, Kirsner and Palmer do not regard such therapy as an additional hazard. Ripstein observed this complication more often with acute fulminating colitis, and reported 12 perforations in 142 operative cases, 8 of which occurred during acute exacerbations. Radiologically, impending perforation is suggested by distention of a segment of bowel which is atonic and fixed in position. Lesser bowel distention also appears. This ileus suggests seepage through the weakened and edematous colon, and should not be mistaken for intestinal obstruction.

RIGHT-SIDED AND REGIONAL COLITIS

In about 8 to 10 per cent of patients with ulcerative colitis, particularly younger peo-

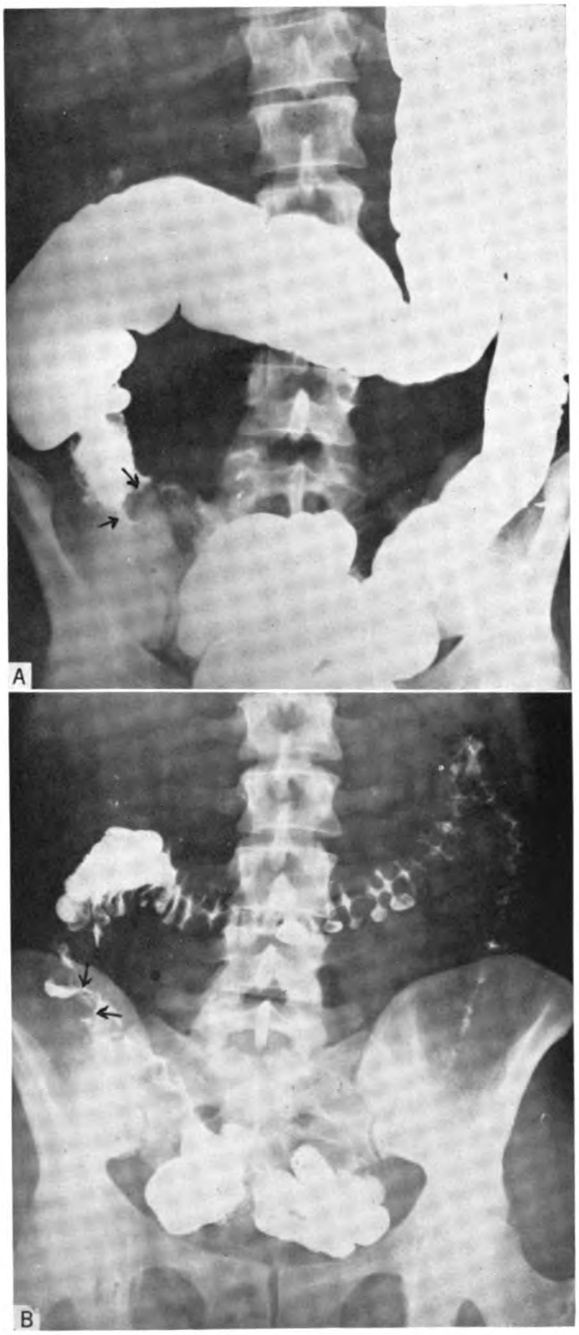

FIG. 146.—*A*, Right-sided colitis in a 36-year-old woman with pain in the right lower quadrant. A tender mass was palpated, diagnosed clinically as an ovarian cyst. Barium enema shows spasm and irregularity of terminal ileum and ascending colon. A circular indentation is present in the caput cecum (arrows).

B, Post-evacuation film, same patient. The irregular mucosal pattern of the terminal ileum and ascending colon are better seen. Note the polypoid shadow in the cecum (arrows). At operation an inflammatory lesion of the terminal ileum and ascending colon diagnosed as benign right-sided ileocolitis was found.

ple, the lesion is situated mainly on the right side, and progresses caudally with intervening skip areas of normal bowel. With the advance of the disease the skip areas merge into the affected colon. Associated with right-sided colitis is involvement of the terminal ileum which bear some resemblance to the changes noted with regional enteritis. Otani pointed out that unusual forms of combined regional enteritis and right-sided ulcerative colitis have been observed, similar deeply ulcerative changes in the ileum, and in the cecum and ileum, suggesting that these two processes have the same origin. Possibly a relationship exists between these two and the common form of ulcerative colitis. Neumann, Bargen and Judd described regional colitis as mucosal ulceration with nonspecific chronic granulomatous thickening of the involved area. The ileal inflammations are identical with those in the colon. These authors suggested that the earliest lesion is a purulent cryptitis, followed by microscopic abscess formation and ulceration of the overlying mucosa. Warren and Sommers described the gross and microscopic changes of diffuse regional enteritis as quite different from ulcerative colitis. Colonic extension of such ileitis is present in about 15 per cent of cases, and this should not be confused with regional segmental ulcerative colitis. The microscopic changes caused by colitis are superficial, involving mainly the mucosa and submucosa. This is contrasted with the extensive granulomatous lymphangitis seen with regional enteritis.

Clinically, right-sided colitis presents less severe symptoms than the usual forms of diffuse ulcerative colitis, and rarely causes the dramatic and toxic picture seen in the advanced types of colitis. Urgency, diarrhea and bleeding are less marked, but fever, joint pains and ocular complications are more frequent. Periods of remission are fairly frequent.

Another form of regional colitis related to right-sided colitis is segmental regional colitis which originates distal to the cecum. These, too, may originate as localized lesions, and later assume the characteristics of separate processes separated by skip areas of normal bowel.

Radiologic investigation of patients with regional colitis discloses a shaggy, roughened irregular bowel wall. Fluoroscopically, considerable irritability of the colon immediately adjacent to the involved areas is present, and often simulates a partial obstruction. Visualization of the terminal ileum may require gastrointestinal examination from above. The jejunum usually is normal, and the proximal ileum may show nothing more than a rather smoothened wall. The distal ileum discloses in classical cases a mild narrowing and smoothening, the terminal portion appearing in some cases as a relatively tubular structure. If this loop is watched, it becomes evident that the tubulation and smoothening is more a manifestation of irritability than an infiltrating process. Fluoroscopic and compression studies reveals these loops to be movable, with changes in their appearance from time to time. No constancy in positions such as may be due to fixation of bowel by serosal inflammation is present.

Eight cases of segmental colitis were observed by us, with lesions limited to the proximal transverse colon, the hepatic flexure, the ascending colon and the sigmoid. In these patients the involved areas were sharply delimited, and the radiologic diagnosis was neoplasm. Most were patients between 40 and 70 years of age, with clinical pictures of pain and abdominal distress and bleeding from the bowel. In 4 an abdominal mass was palpated. Inspection of the specimen in 4 cases disclosed concomitant localized adenomatous polyposis. The true nature of the lesion was disclosed by histologic examination.

We also observed 4 cases which might be classified as right-sided colitis, with involvement of the cecum and ascending colon, and associated narrowing of the terminal ileum. These occurred in patients between 10 and 20 years of age, and were associated with a less severe clinical picture. Radiologically, the irritability of the ascending colon was pronounced. Extension into the terminal ileum could be identified in 2 cases, the extent of change involving the terminal 15 or 20 cm.

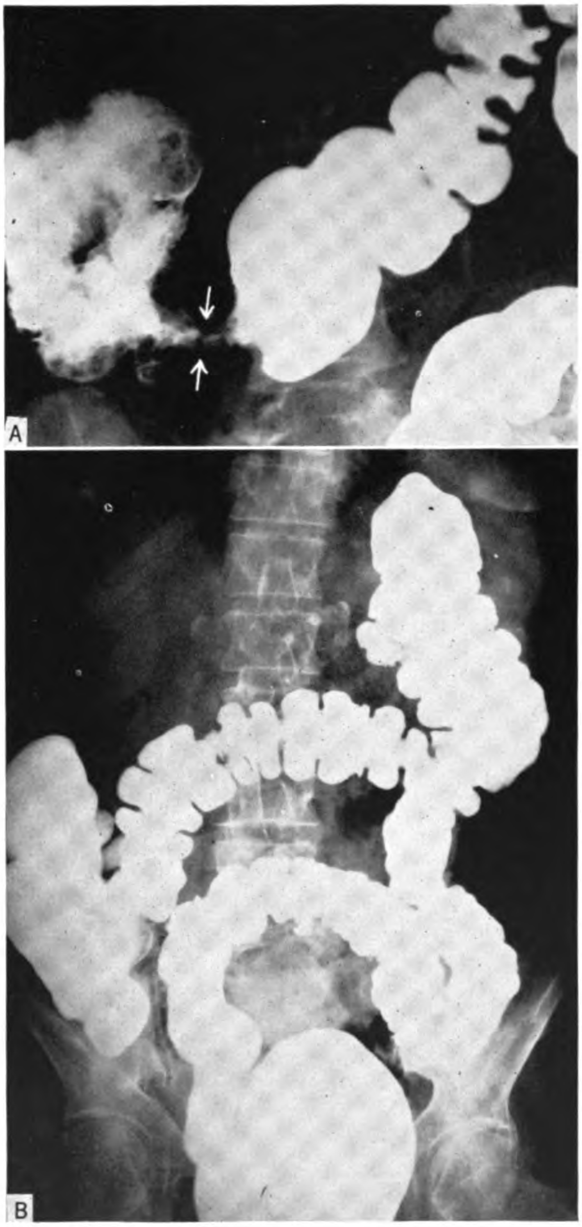

FIG. 147.—*A*, Benign regional colitis in proximal portion of transverse colon in a 70-year-old man with repeated bleeding from the colon. Spot roentgenogram reveals an annular lesion (arrows) interpreted as due to malignant disease.

B, Same patient. The lesion is hidden behind the filled ascending colon. At operation a benign ulcerating lesion was found. Histologic examination disclosed a benign ulcerating regional colitis.

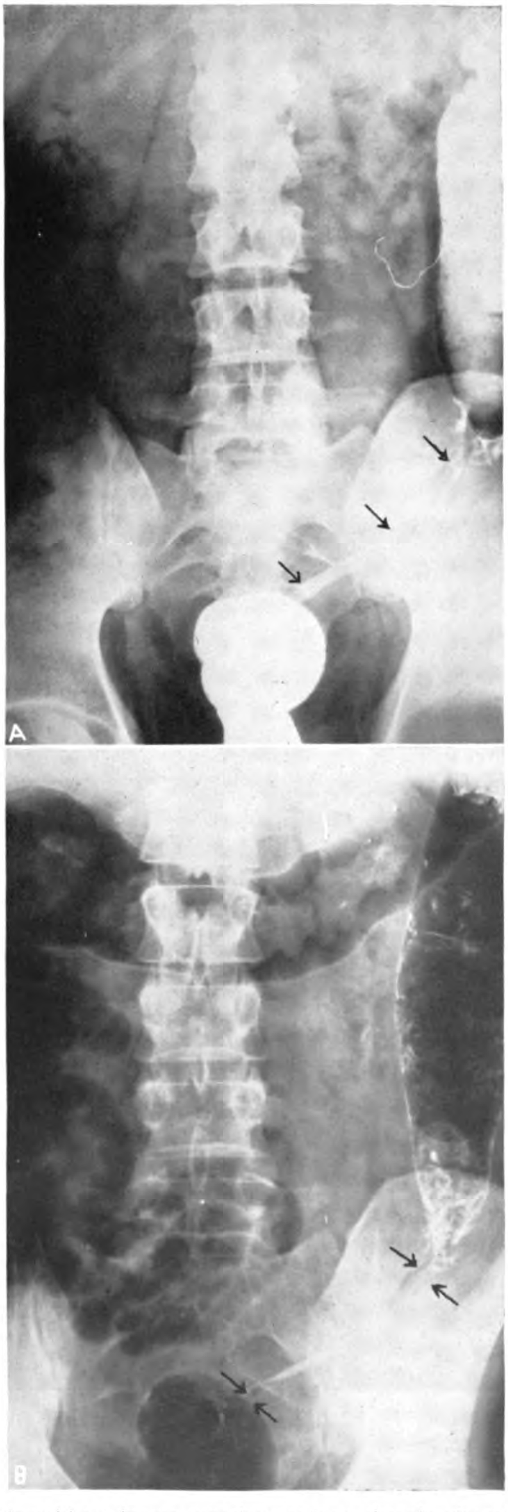

FIG. 148.—*A*, Elongated sigmoid carcinoma superimposed on chronic ulcerative colitis in a 25-year-old woman with bloody diarrhea for 10 years. Pain and tenderness in left lower quadrant, with palpable mass. The colitis symptoms had subsided considerably when this was found. Barium enema shows partial block. The neoplasm is more than 15 cm. long (arrows).

B, Air contrast examination, same patient. The bowel above the neoplasm is distensible, and pseudopolypi are present. At operation a large carcinoma with peritoneal metastases was found.

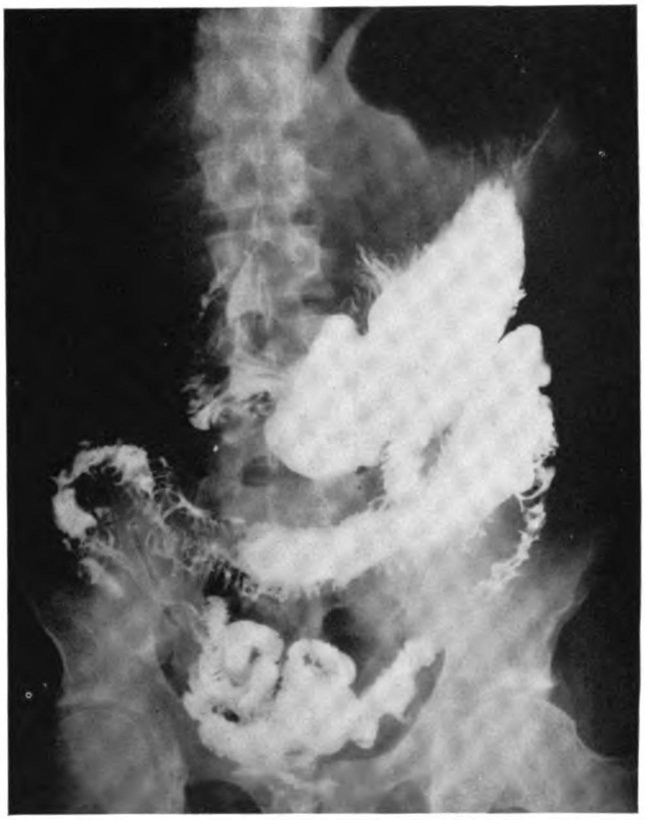

FIG. 149.—Cologastric fistula from transverse colon to greater curvature in a 30-year-old man with ulcerative colitis for 2 years.

CARCINOMATOUS DEGENERATION IN ULCERATIVE COLITIS

Malignant degeneration in ulcerative colitis has been reported in about 4 to 5 per cent of cases. Usually it is a fairly late complication, but it appears in any age group. Neoplastic degeneration begins insidiously, and the diagnosis is best established if patients who are seemingly improved are subjected to constant check. The disease appears mainly in patients with diffuse ulcerative colitis. It has not been observed with regional enterocolitis. Neoplastic infiltration appears approximately 15 years after the onset of the disease. In our experience carcinomatous degeneration was mostly in the transverse and sigmoid colon. In 5 of the 8 cases observed, the lesion was long and had smooth walls. No gross obstruction was present, the lesions encircling the bowel. It is of interest that frequently the tumor was found on routine check examinations.

Enterocolic or cologastric fistula formation is a rare complication of ulcerative colitis. One such case was seen by us in a 30-year-old man who had had colitis for 2 years. The diagnosis was made during barium enema examination, when barium was observed entering the stomach. Examination of the upper gastrointestinal tract showed no direct passage of barium from the stomach to the colon. At operation the colon and stomach were adherent, with multiple smooth-walled, sharply definite ulcers in the stomach and colon. No large intervening mass or abscess was present. Another such case was mentioned by Rowe. In his patient the cologastric fistula healed spontaneously, and could not be demonstrated at operation 3 years later.

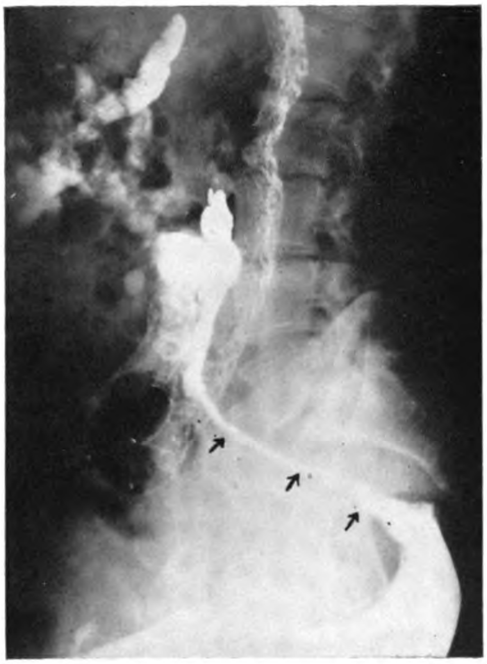

FIG. 150.—Perisigmoiditis due to peritonitis in a 45-year-old male operated upon for a perforated duodenal ulcer. Sigmoidoscopy revealed mucosal edema. A boggy, slightly tender mass was felt in the *cul de sac*. Barium enema shows marked narrowing of the sigmoid (arrows). Patient recovered with antibiotic treatment.

PERICOLITIS

Serosal inflammatory changes may reflect disease of adjacent organs. These are seen most often in the distal third of the colon, particularly the perisigmoid and perirectal regions. Adnexal diseases, particularly pyohydrosalpinx and pelvic abscesses, involve the adjacent bowel in such a fashion that malignant disease is diagnosed. Incidentally, serosal implants of cancerous tissue may cause a very similar appearance. In some, an irregular narrowing of the colon appears, while in others the involved wall becomes a diffusely smoothened tube. The diagnosis in most instances is facilitated by due regard to the history and the changes detected on physical examination.

Occasionally a patient with peritonitis presents a somewhat similar picture, particularly when a pelvic cellulitis is part of the disease. In 1 of our patients this followed a subtotal gastrectomy. Radiologically the sigmoid was tubular and thin, with smooth walls. On proctosigmoidoscopic examination only reddening and edematous mucous membrane was visible, out of line with the deformities noted radiologically. This disparity served as a clue to the inflammatory nature of the lesion, which subsided on medical treatment. In 2 other patients who were operated upon with a clinical diagnosis of malignant disease or diverticulitis without visible diverticula, the wall of the bowel was found to be indurated and friable, while the serosal surfaces contained necrotic material.

Pericolitis is known to follow perforation of the bowel by ingested foreign bodies. In these patients a diffuse infiltration surrounds the involved area, which may be quite elongated.

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DIVERTICULITIS

Congenital diverticula of the stomach and lesser bowel were discussed previously. The complications of perforation or acute inflammation gives rise to acute symptoms including pneumoperitoneum, peritonitis, bleeding or intussusception.

For the most part, diverticulitis occurs in the large intestine, particularly the descending and sigmoid colon. Diverticulosis is considered an acquired degenerative disorder, with weakening of the muscular walls and protrusion of mucosa between the separated muscle fibers. Diverticula thereby are sacs formed by mucosa, submucosa and serosa, and usually they are situated in multiple rows along the convexity of the colon rather than along the concavity of the loop as seen in the small intestine. In many cases diverticula are arranged in two rows along the edges of the longitudinal bands of the large intestine. Diverticula are found anywhere in the large bowel above the peritoneal reflection, and have also been seen in the appendix. They vary in size from those so small as to escape detection to others measuring up to about 4 cm. in diameter. Most do not exceed 1 to 1.5 cm. in diameter, and mainly are sessile structures, but occasionally some have necks. The openings vary in size from pinholes to those which readily admit a finger tip.

Diverticula limited to the right colon are relatively uncommon. Considerable importance is attached to solitary diverticula

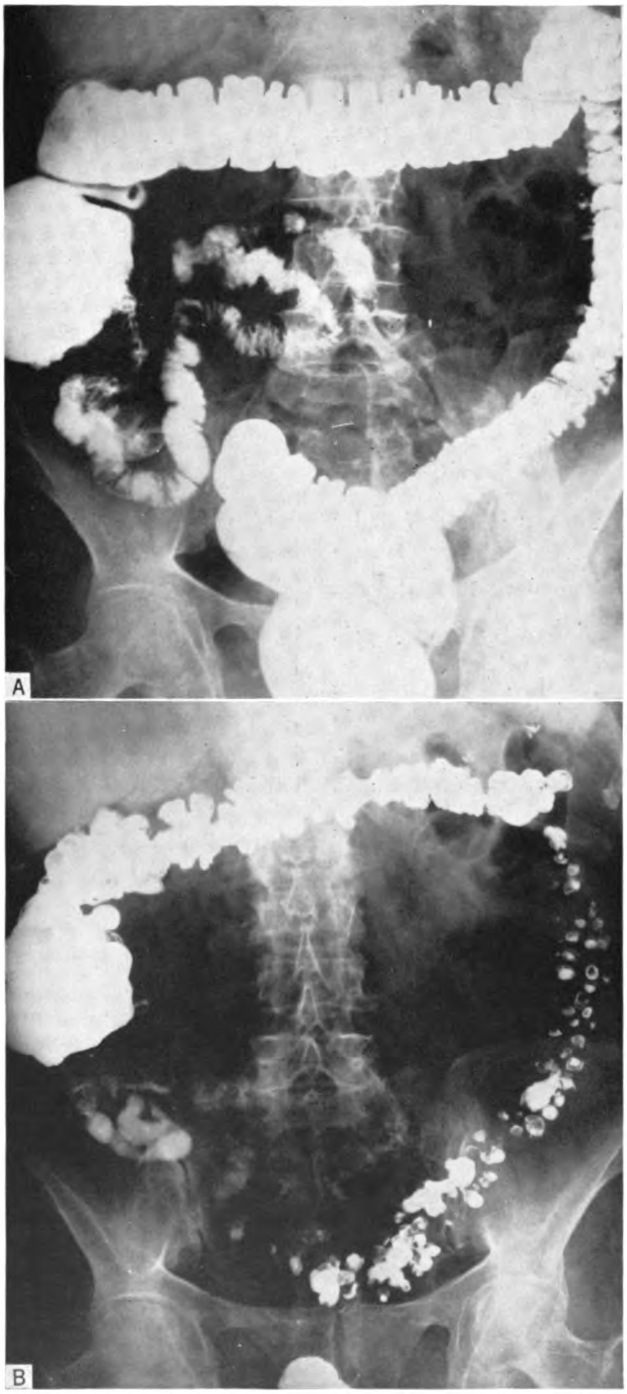

FIG. 151.—*A*, Barium enema film of a 69-year-old woman with diverticulosis of the descending and sigmoid colon.

B, Same patient, post-evacuation film. The diverticula are better demonstrated.

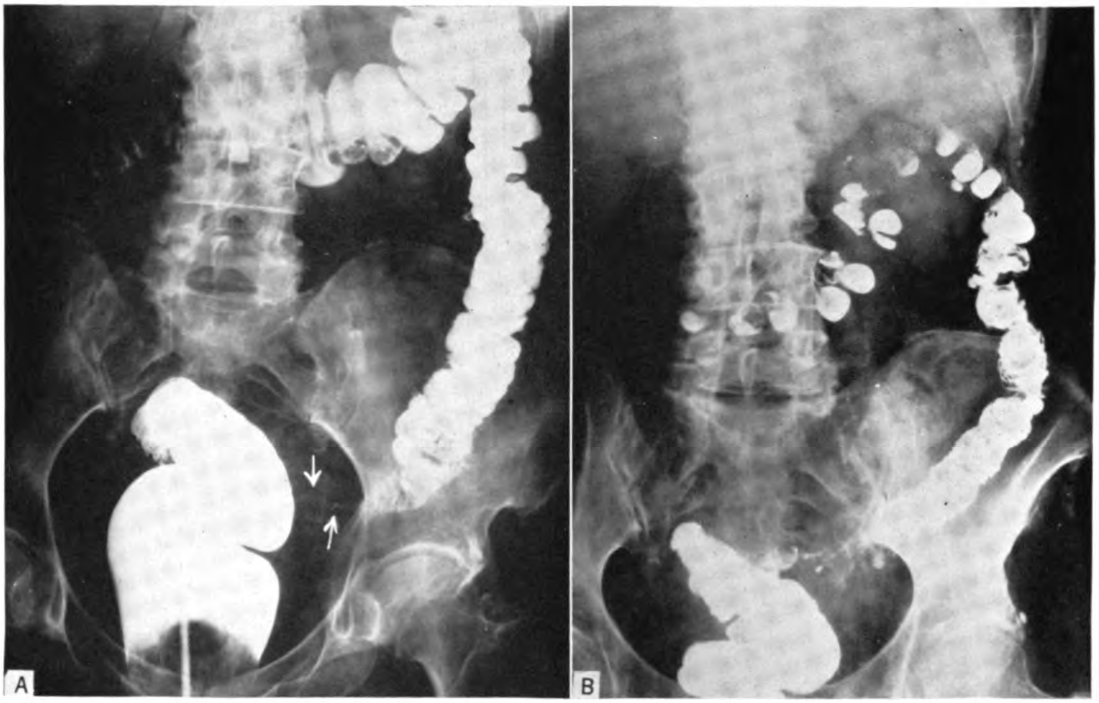

FIG. 152.—*A*, Barium enema reveals a partial block due to diverticulitis in the sigmoid (arrows) in a 58-year-old woman with lower abdominal pain, weight loss and change in bowel habits. Note the tapered distal descending colon.

B, Same patient, post-evacuation film. A tiny diverticulum is present. The mucosal pattern is irregular. At operation it was felt that the patient had a malignant lesion, but on cutting the specimen purulent droplets exuded. Further microscopic examination revealed diverticulitis.

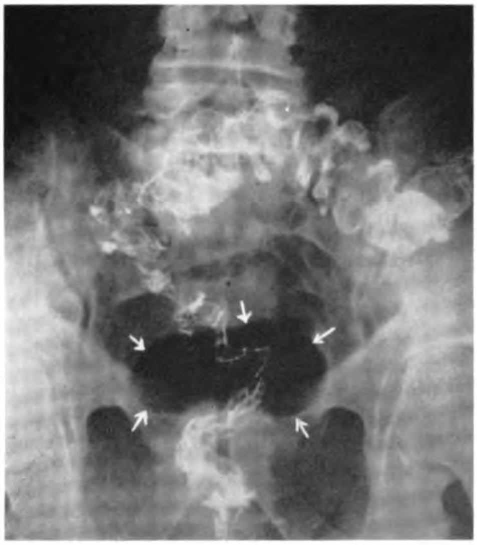

FIG. 153.—Rectovesical fistula in a 54-year-old man with diverticulitis. Note the air in the urinary bladder (arrows).

of the cecum and ascending colon. These patients clinically simulate acute appendicitis, and with chronic inflammation, resemble carcinoma of the cecum or some granulomatous disease. With pronounced inflammation is present, extension to the serosa of the ileum is sometimes observed, simulating regional enteritis.

Colonic diverticula are relatively uncommon before 30 years, and their incidence increases with the advancing years. Males are affected about twice as often as females. It has been estimated that from 5 to 10 per cent of the population over 40 years of age has diverticulosis, and that from 15 to 20 per cent of these later develop diverticulitis. In the early stage, diverticulosis may present as nothing more than a slight irregularity along the wall of the bowel, referred to as pre-diverticulosis. Even when well-formed, uncomplicated diverticulosis is not accompanied by symptoms. However, when in-

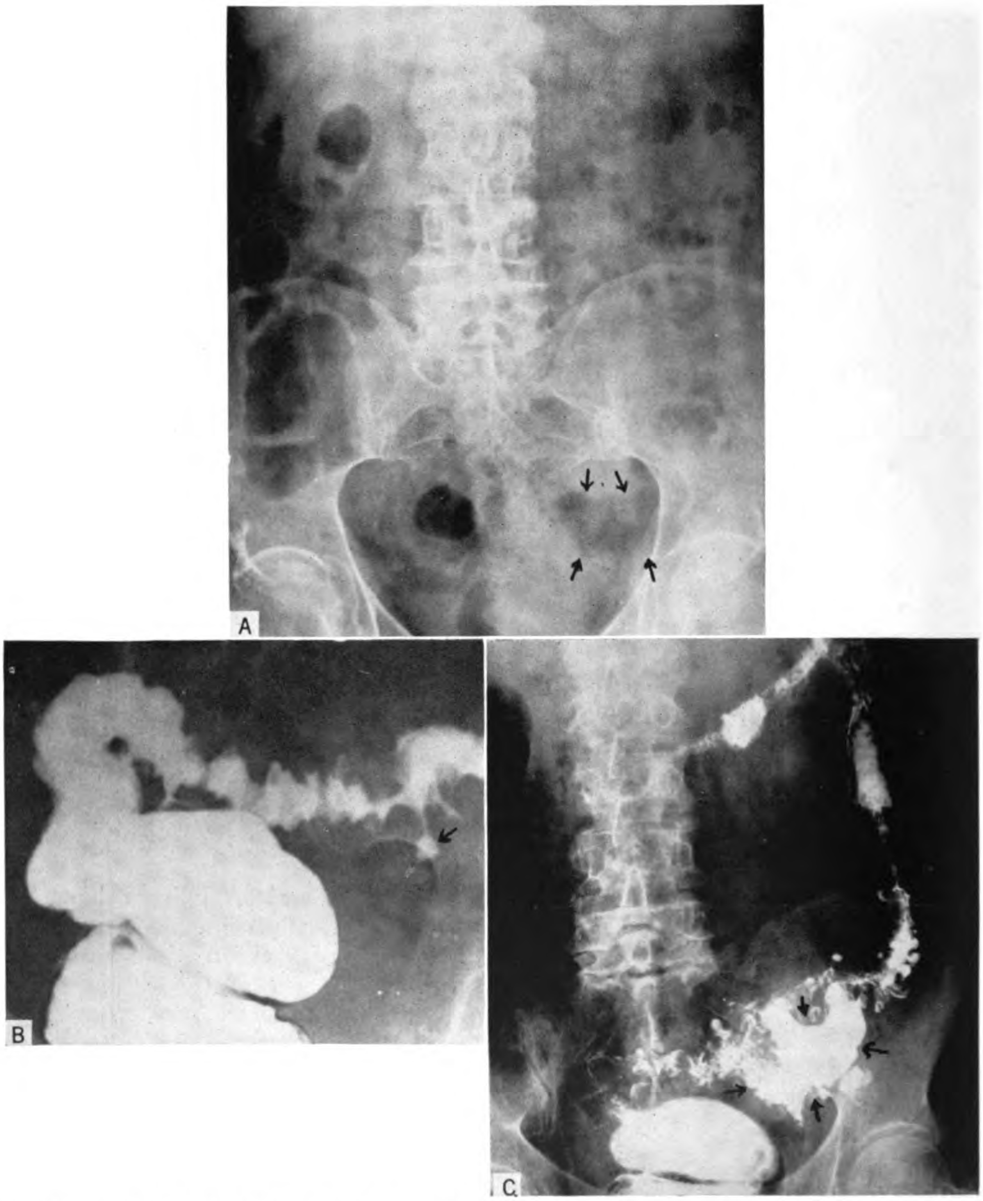

FIG. 154.—*A*, Perforating diverticulitis in a 79-year-old woman with vomiting and abdominal pain for two weeks. Mass felt in left lower quadrant. Scout film shows local collection of gas in the left lower quadrant (arrows).

B, Spot film made during barium enema shows barium occupies the area of the gas collection seen in Figure 154*A* (arrows). Numerous diverticula are present in the sigmoid.

fection supervenes, a considerable degree of clinical significance is attendant. So long as diverticula are open and uninflamed, their contents vary from inspissated stool to relatively fluid material. The emptying capacity of diverticula is indicated by the variable time barium persists in them. While as diverticular contents can empty into the lumen of the bowel, no clinical disturbances appear. However, should they fill with hard stool, sufficient pressure change can be produced to cause rupture of the mucosa, fol-

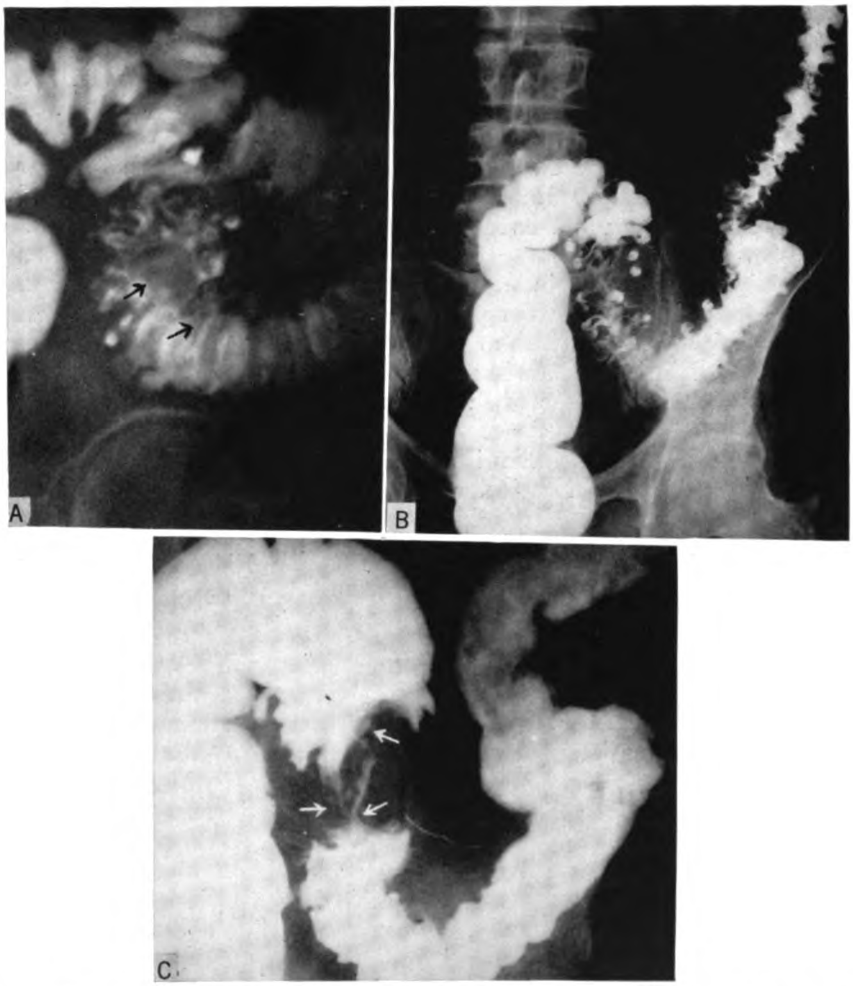

FIG. 155.—*A*, Spot film roentgenogram in a 63-year-old man with recurrent abdominal pain and rectal bleeding for 10 years. A pedunculated polyp is present in the sigmoid (arrows).

B, Same patient, 1 year later. Numerous diverticula are present in the descending and sigmoid colon. A delay was noted at the sigmoid during fluoroscopy.

C, Same patient, 1 year after Figure 155*B*. A neoplasm is present in the sigmoid (arrows).

lowed by inflammation. This may be purely local and asymptomatic. However, relatively acute inflammation gives rise to symptoms of pain and tenderness, and a mass may appear if the peridiverticular inflammation becomes intense. Acute diverticulitis is accompanied by fever and chills, cramps, changes in bowel habits, and other systemic manifestations. Abscess formation about the bowel causes partial or even complete obstruction, and further extension of such suppuration is responsible for a wide variety of fistulas. These are present in from 10 to 15 per cent of patients coming to surgery, and extend from the colon to the bladder, the vagina, ureters and the retroperitoneal region. Fistulas leading to the stomach have been observed rarely.

The clinical picture of the various fistulas reflects the area involved. With bladder fistulas the characteristic complaints are frequency, dysuria, and passage of gas and stool with the urine. Occasionally hematuria is an important symptom. Vesicosigmoid fistulas are more common in males. In females the uterus is believed to exert a protective influence by walling off the inflammatory changes. Retroperitoneal fistulas are less frequent, and present a more complex picture. Unexplained pain and fever sometimes point towards such a tract, and the diagnosis is established by radiologic visualization of contrast material entering the involved area.

The location of perforation also influences the clinical picture. Not uncommonly a patient with a perforated diverticulum of the sigmoid has complaints referable to the right lower quadrant. This occurs when a relatively long loop of sigmoid lies there.

Infrequently lesser bowel obstruction is a presenting symptom. Here the diverticulitis catches a loop of small intestine, producing mechanical obstruction by adhesions kinking the affected bowel.

The occurrence of bleeding with diverticulosis and diverticulitis is still a subject of discussion. Rushford has demonstrated by careful dissection that sizable blood vessels are present in the vicinity of diverticula. It is through the weak spots alongside these that the original protrusions arise. He found that bleeding has been observed with diverticulosis in about 17 per cent of cases, computing his figure from 45 reported series totaling 6,000 patients. Hemorrhage varies with the size of the vessel involved and the site of the lesion. Melena may be produced by proximal colonic diverticulitis, and massive hemorrhage has been observed in patients unsuspected of diverticulitis. He noted that blood has been seen coming from diverticular orifices on sigmoidoscopy or after bowel resection. Bleeding appeared in 27 per cent of his 120 cases, the quantity of blood lost varying from a trace to exsanguinating hemorrhage. In a series of 317 unselected cases, Keith and Rini noted that 19 had rectal bleeding or massive hemorrhage. On followup studies of these over periods of from 4 to 10 years, there were 10 survivals. Of these, 8 had no recurrent bleeding. Two bled again, and on re-evaluation no other lesion was demonstrated. According to these authors, no patient has been known to die of hemorrhage due to diverticulitis.

Other opinions are at variance with these two authorities. Kitchen suggested that bleeding from diverticula be accepted only with reservation. Our experiences are in accord with this opinion. Bleeding should not be attributed to diverticula until a thorough search for other causes has been made. The association of lesions such as polyps and tumors must not be forgotten in the presence of even extensive diverticulitis, which often serves to obscure a more important lesion. Radiologic examination of patients suspected of tumorous conditions are more difficult when diverticula are present. Here the identification of a pedicle on compression studies may be of fundamental importance. Another point sometimes of help is the taking of 24- or 48-hour films. Barium retained within a diverticulum sometimes settles the question as to whether a shadow under scrutiny is a polyp or diverticulum.

The radiologic diagnosis of diverticulosis is established either by barium enema or upper gastrointestinal examination, preferably the former. Occasionally scout films suggest the presence of diverticulosis and even of diverticulitis. Massik and Wheately saw 12 such cases in 370 examinations. We were able to identify 25 similar cases. In three diverticular pockets of gas were present in the left lower quadrant close to several similar larger collections. These represented communicating abscesses, verified by entrance of barium on colon examination. The appearance of diverticulosis as demonstrated on barium enema is so well known that further discussion is not necessary here. The identification of diverticulitis, however, requires considerable acumen, especially in the early stages when only irritability and spasm of the bowel suggests in a most indefinite way the presence of inflammation. Such deformities are transient, and often are rather extensive. However, it is not too uncommon to find a relatively localized lesion which cannot be differentiated from a malignant mass. This may require repeated examinations, but if there is any doubt operation should be advised. Indeed, the presence of a localized diverticulitis in itself is considered an indication for surgery in many patients.

During the acute phase of diverticulitis, sufficient spasm to produce partial obstruction is encountered occasionally. This may be indicated during fluoroscopy while a barium enema is being introduced by an initial ballooning of the rectum, followed by entrance of barium into the colon proximal to the spastic area. These patients usually do not have symptoms of bowel obstruction, and in most cases repeat examinations within a week after treatment shows improvement in the flow with relatively little distortion of the bowel. However, when the bowel wall is infiltrated and inflamed, complete obstruction with the attendant clinical signs may appear. These, too, may subside with proper medical care, but often come to surgery because of persistent symptoms of pain and discomfort.

Acute diverticulitis sometimes appears when there is no radiologic evidence of concomitant diverticulosis. The lesion is then identified only after the removed organ is dissected and subserosal globules of pus extending towards the bowel lumen are found. Indeed, the surgeon and pathologist often cannot tell whether or not the excised loop of intestine is cancerous by inspection alone.

As a rule perforation of diverticula is walled off by the surrounding inflammation. Occasionally perforation directly into the peritoneal cavity produces pneumoperitoneum and peritonitis. The possibility of perforating diverticula in the course of barium enema examination is rather small, but 4 such cases were seen by us. In one there was a concomitant neoplasm of the sigmoid proximal to the perforated diverticulum.

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EPIPLOPERICOLITIS

The wall of the bowel also may be inflamed by infection originating in the appendices epiploica. This condition also is referred to as tumoral epiploitis, inflammatory pseudotumor of the colon, epiplopericolitis, granuloma of the colon or Braun's tumor. Perforation of the bowel by a foreign body such as a fishbone, and occasionally cholecystitis, are considered underlying causes.

The epiploic appendages are fat-filled serosal covered appendages occurring on the large intestine, including the appendix but excluding the rectum. The enveloped fat is a continuation of the subserosal fat of the intestinal wall, and is related to the longitudinal bands of the colon. These appendages are arranged in two rows. Their average length is about 3 cm., but they have been observed to be as long as 15 cm. Diverticula in the substance of the epiploic appendages are due to their proximity to the sites where the vessels enter the intestinal wall. When acutely inflamed the appendices epiploica can undergo infarction, gangrene and suppuration, simulating an acute abdominal disorder. In more chronic stages they may become calcareous. When the inflammation extends around the bowel symptoms of obstruction develop.

Radiologically, epiplopericolitis is associated with obstructive changes of varying degree. On barium enema examination deformity of the bowel may appear as a funnel-shaped narrowing between two cone-shaped extremities, assumed to be due to extrinsic pressure against the mucous membrane. The extent of the lesion usually is longer than that customarily observed with carcinoma, but this is an unreliable sign. Di Rienzo and Mosca suggested that the injection of 1 c. cm. of pitressin is helpful. This causes frank activity of the muscularis mucosa, so that peristalsis becomes visible during fluoroscopy.

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MESENTERIC ADENITIS

One of the causes of acute abdominal pain in children is sudden nonspecific inflammation of the mesenteric lymph nodes, frequently associated with upper respiratory infections. Abdominal pain often simulates acute appendicitis, and in older patients renal colic or gastrointestinal disturbances of diverse origin are mimicked. Radiologic examination of the abdomen fails to disclose any characteristic change. Considerable ileus may be present.

Acute mesenteric adenitis infrequently is the predisposing cause of acute chylous peritonitis in infants and young adults. The rarity of this is indicated by the report of Hoffman, who described a case of acute chylous ascites with free chyle in the peritoneal cavity, presumably the second case due to acute mesenteric lymphadenitis. In this patient, a 10-day-old infant, the abdomen was distended and free fluid was obtained when the abdomen was opened for possible acute intestinal obstruction. Multiple large mesenteric lymph nodes were present. This condition should be separated from the more frequent chronic chylous ascites.

A case of acute chylous peritonitis in a 27-year-old man due to tuberculous obstruction and erosion of the lumbar lymphatic ducts was reported by Walker. Radiographic examination of the abdomen failed to help in the diagnosis.

Another rare complication of acute mesenteric adenitis is primary mesenteric abscess. This condition was discussed by Dudley and MacLaren, who reported 2 cases presumably due to suppuration of mesenteric lymph nodes. One was a 42-year-old male in whom the roentgenograms suggested an incomplete obstruction. The second, a 32-year-old man, had no *x*-ray examination, the condition having been diagnosed as an appendiceal abscess. These authors found 7 published cases of solitary lymph nodes abscesses, and also mentioned examples of diffuse suppurative mesenteric adenitis in which small foci of suppuration and enlarged lymph nodes were seen at operation or at necropsy as part of a generalized peritonitis.

The association of ileocolic intussusception

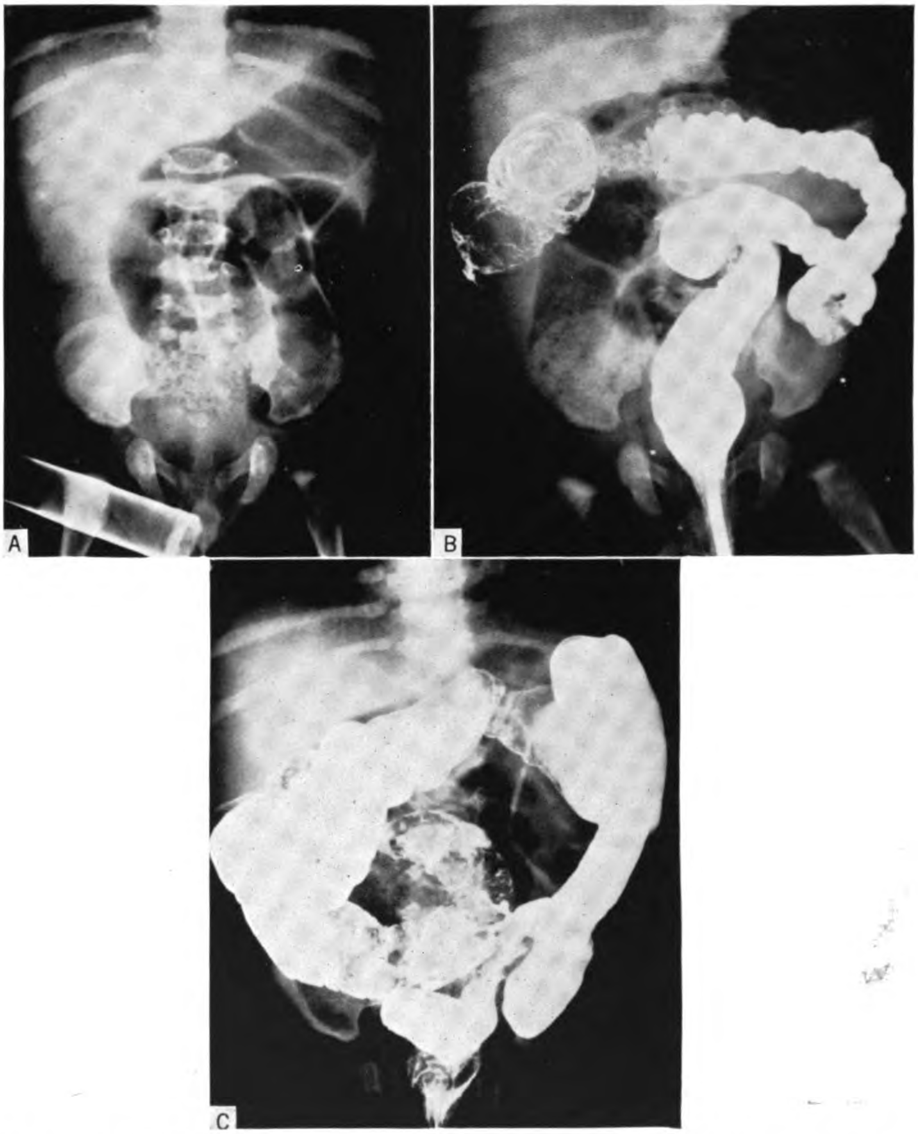

FIG. 156.—*A*, Scout abdominal film of a 3-month-old male infant reveals marked distention of the lesser bowel. No gas is present in the right upper abdomen. This is highly suggestive of lesser bowel obstruction.

B, Barium enema reveals a coiled spring mass in the right upper quadrant indicative of intussusception. The lesser bowel distention is pronounced because of ileo-ileocolic intussusception.

C, Same patient. The ileocolic intussusception has been reduced, and barium is present in the distended lesser bowel. Ileoileal intussusception was found at operation.

with enlarged lymph nodes at the ileocecal junction is worthy of comment. It has been suggested that these nodes may be a factor of some importance. Hogg and Donovan indicated that this might be part of a generalized lymphatic hypertrophy involving the terminal ileum as well as the adjoining mesenteric nodes, and attributed intussusception to this lymphadenopathy. They encountered this situation in 11 per cent of their cases. The presence of hypertrophy of Peyer's patches in the terminal ileum with

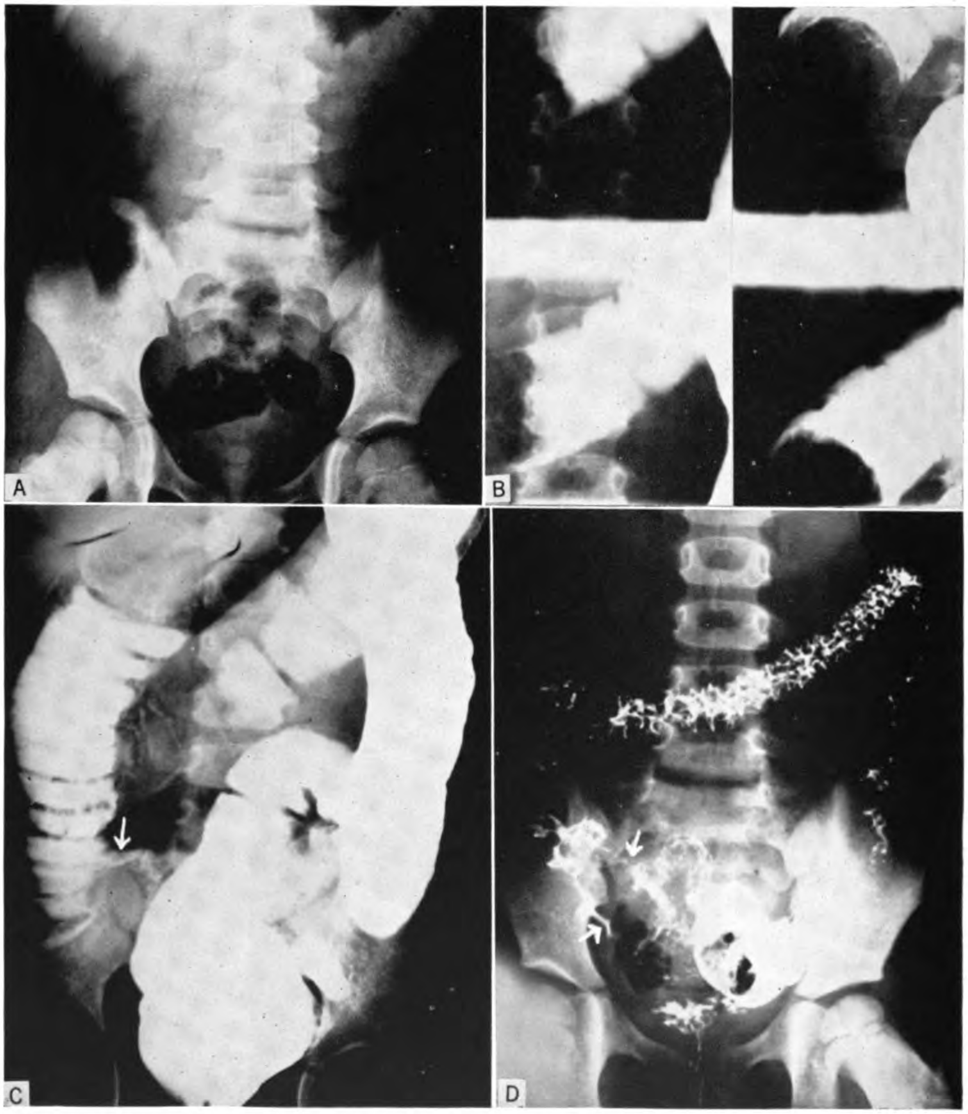

FIG. 157.—*A*, Scout film of a 3-year-old girl with cramps and lethargy. Occult blood in stool. Note the absence of gas in the right upper quadrant. A dilated loop of lesser bowel is in the pelvis. This is highly suggestive of intussusception.

B, A series of spot films made as barium meets the head of the intussusception.

C, Same patient. The ileocolic intussusception has been reduced, and barium enters the appendix and terminal ileum (arrows).

D, Post-evacuation film. The terminal ileum and appendix are still visible (arrows). Patient made an uneventful recovery.

intussusception was commented upon by Sarason, Prior and Prowda, who reported a patient in whom 5 episodes of recurrent ileocolic intussusception was found to have been caused by a tumorous mass in the terminal ileum. On microscopic examination this presented characteristics of giant follicle hypertrophy of Peyer's patches. Hypertrophied mesenteric lymph nodes with enlarged lymphoid follicles and spasm of the adjacent bowel was regarded by Orloff as a likely cause of intussusception in children.

Suffice it to say at this point that there is no generally accepted theory for the mechanism of this condition.

In 6 of our cases operation disclosed enlargement of the lymph nodes in the ileocecal angle with intussusception. In 1, the removed node disclosed only diffuse lymphadenitis. Operation had been undertaken in these patients because hydrostatic reduction of the intussusception was regarded as incomplete. In 1, a concomitant ileoileal intussusception had been suspected because of a persistent dilatation of the lesser bowel, and this was verified at operation. In 4 others, no intussusception was found at operation, and in the sixth a recurrent intussusception was present up to the transverse colon. The question as to what indicates adequate hydrostatic reduction is a disputed one. In the 4 cases mentioned above, the contrast medium did not enter the terminal ileal coils, but the caput cecum was distended, and in 2 the appendix was visualized. While we much prefer to see the terminal ileum fill when hydrostatic reduction of intussusception is done, the cases cited indicate that absence of such filling does not in itself indicate failure of the procedure. In similar cases since then we have completed the study by giving the patient from 20 to 40 cc. of water-soluble contrast material by mouth. This flows quite rapidly through the intestinal tract, and if seen in the colon within an hour is regarded as indicative of resolution of the intussusception.

Chronic mesenteric adenitis most frequently is due to tuberculosis. No definite symptoms are produced in most patients, and the diagnosis often is made because of calcification seen within lymph nodes on scout films. Other diseases such as brucellosis and tularemia infrequently cause chronic mesenteric lymphadenitis, and histoplasmosis, coccidiomycosis and blastomycosis also cause this condition. Enlargement of intraabdominal lymph nodes secondary to Boeck's sarcoid is seen occasionally. This produces no characteristic radiologic change.

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PERITONITIS

The peritoneal cavity is lined with flattened mesothelial cells supported by connective tissue. When irritated these proliferate and become cuboidal. A vast surface is provided both for exudation and absorption of fluid. The omentum, which is part of the peritoneal surface, appreciably increases this area. The mobility of the omentum towards inflamed areas has been recognized by the term "policeman of the abdomen." The pleural and peritoneal cavities are in potential contact with one another because the peritoneal surface of the diaphragm is studded with small pits into which the peritoneum dips. This permits close approximation between the lymphatics of the diaphragm and those of the thoracic and peritoneal surfaces.

The onset of intraperitoneal inflammation is followed by a diffuse or localized reaction, often with exudation of fluid. In most cases peritonitis is caused by infected material from involved viscera which may rupture either suddenly or gradually. Trauma or direct penetrating wounds give rise to a similar process. Less often infection is hematogenous in origin, as may occur with pneumococcal peritonitis which produces an acute picture with rapid exudation of fluid and ileus. Peritoneal infections sometimes become localized, or form communicating pockets. Excessive dilatation of the bowel accompanied by increased permeability of its wall is another possible producing cause of peritonitis.

Acute peritonitis often has an overwhelming infection as its outstanding characteristic, and the ileus accompanying this may present radiologic changes easily misinterpreted as intestinal obstruction. Similar changes are associated with acute necrotizing enterocolitis, with a micrococcus as the offending organism. Recently we observed 2 patients in whom a similar clinical picture was due to fungus infection.

In more chronic forms of peritonitis fluid may collect in the lesser sac, or accumulate in intra-abdominal pockets. As a result of healing, fibrous adhesions are formed. These may be completely asymptomatic, or may produce obstructive changes. In some the onset of symptoms is delayed for years. If the adhesions follow surgery, obstructive symptoms may come on relatively rapidly. The differential diagnosis between partial intestinal obstruction due to adhesions and paralytic ileus may be difficult. Paralytic ileus supposedly causes a silent abdomen, with absence of peristaltic sounds. Mechanical ileus is accompanied by hyperperistalsis with increased bowel sounds and cramps. A combination of both processes often exist simultaneously, so that the diagnosis between physiological and mechanical ileus sometimes is not possible. In such cases we have found it useful to do a survey examination with water-soluble contrast materials. If this passes into the colon within 2 hours, the chances are that the block is not mechanical and that it is safe to proceed with a more definitive examination with barium. The interpretation of water-soluble contrast examinations of the upper gastrointestinal tract requires a concept of the normal picture to avoid the mistake of reading partial intestinal obstructive changes as within normal limits.

The radiologic picture of ileus is deceptive and variable. In some nothing more than an occasional distended loop of bowel is seen, sometimes with scattered distended loops of large intestine. With more advanced ileus the entire lesser bowel may be prominently distended, with exaggerated valvulæ conniventes and fluid levels. Welch has pointed out that the combination of intestinal obstruction and peritonitis is a dangerous one immediately postoperatively. While most patients respond to conservative treatment, others who fail to improve require surgery to relieve obstruction, evacuate peritoneal abscesses and to retain as large an absorptive area of intestine as possible. Dolan indicated that hypoproteinemia and edema are frequent complications of mechanical ileus, and may be responsible for superimposed paralytic ileus. The effects of adhesions appear to be more than merely mechanical pressure or angulation. Dolan postulated a localized disturbance in motor physiology at the site of an adhesion manifested by narrowing of the bowel lumen which sometimes persists even after death. The mechanism for this is not clear.

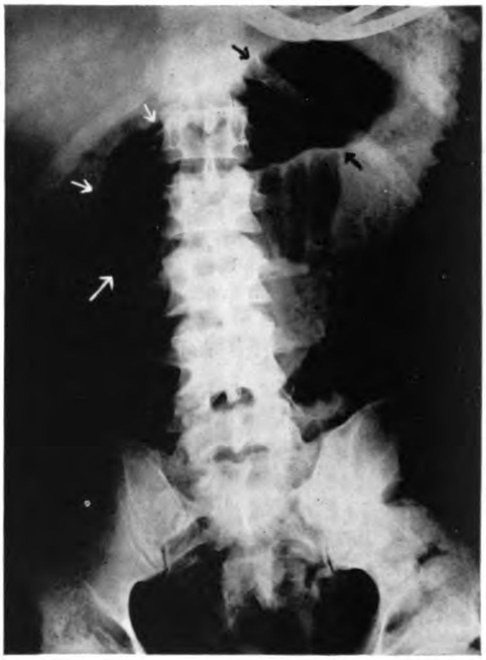

FIG. 158.—Scout film of abdomen in 36-year-old man secondary to adhesions binding down the mid-jejunum, with rotation of the bowel. Marked dilatation of jejunum (arrows), with remainder of bowel relatively collapsed.

In a survey of 50 cases of mechanical intestinal obstruction due to peritoneal adhesions, 5 occurred within 2 to 5 days after operation. This presents a question as to

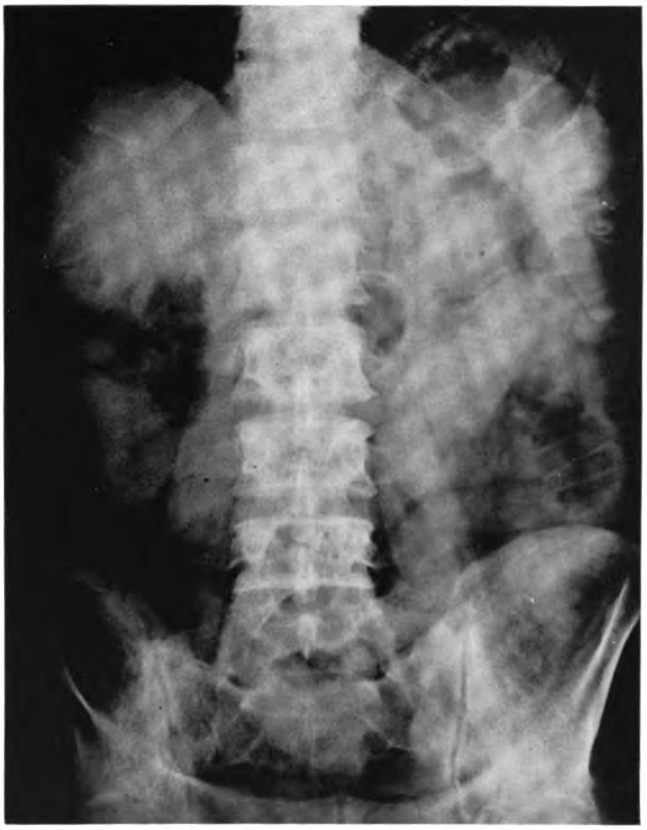

FIG. 159.—Fluid-filled distended loops of jejunum and ileum. Intestinal obstruction due to adhesive band in the right lower quadrant, confirmed at operation. Patient was a 63-year-old male with a history of constipation for 6 days.

whether immediate surgery is required, often a difficult one to answer. The wide variety of lesser bowel distention patterns is impressive, and it seems inescapable to conclude that the diagnosis often is in doubt, and that examinations with contrast materials is preferable to relying upon observation of the gas-distended bowel loops. Obstructive bands form anywhere in the peritoneal cavity, mostly in the pelvis because of the frequency of operations in that area such as appendectomies, hysterectomies and hernia repairs. A loop of bowel sometimes rotates about a band, resulting in obstructive volvulus with vascular impairment. These may present relatively acute and rapidly progressive abdominal symptoms. If seen in early stages, the rotated and distended loop of intestine may not contain gas, and sometimes will resemble a mass lesion. If the wall of the bowel between the apposing segments is thickened and edematous, a band between them may be observed. If gas enters the involved intestine, a hair-pin configuration of a radiolucent nature appears. Solis-Cohen and Levine pointed out that an early sign of mechanical obstruction of the small intestine may be heralded within 2 to 4 hours after the onset of symptoms by a similar hair-pin turn, and regard this as a forerunner of the later step-ladder pattern of distended transversely situated loops with prominent mucosal folds and fluid levels. The absence of fluid levels does not exclude intestinal obstruction. In some instances fluid-filled intestinal coils are visualized on plain films by virtue of their increased density.

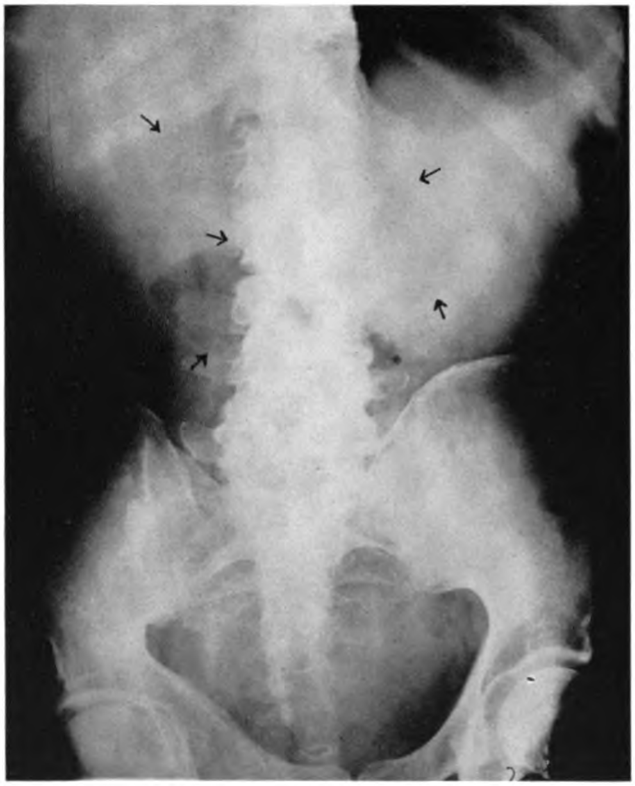

FIG. 160.—Closed loop obstruction of ileum in a 60-year-old man with coffee bean sign (arrows) in epigastrium. Volvulus and infarction of lower 4 feet of ileum about old adhesions found at operation.

Ileus due to other acute disorders may mimic intestinal obstruction both clinically and radiologically. A patient with a ureteral colic, for instance, can present a picture indistinguishable from intestinal obstruction unless the diagnosis is considered. Similar difficulties arise with acute ulcers, particularly with bleeding. Other conditions in which ileus is prominent include such diverse entities as acute hemorrhagic pancreatitis, acute cholecystitis, pneumonia, uremia, severe blunt abdominal trauma and the ileus attendant on collagen diseases and various medications.

Intra-abdominal fluid is fairly frequent with peritonitis. Separation of distended loops of bowel by relatively radiopaque zones is considered suggestive of fluid between these loops. This was checked by comparing such roentgenographic changes with observations at necropsy, and we are unable to establish a definite correlation. It is equally probable, and perhaps more likely, that these zones are due to edema of the bowel wall.

The differential diagnosis between intra-abdominal adhesions and peritoneal carcinosis often cannot be made without due recognition of the clinical history. Occasionally a careful pelvic examination discloses an ovarian mass in a patient suspected of acute lesser bowel obstruction. Gastrointestinal examination is useful in disclosing linear areas of partial obstruction, which often are multiple. The need for relief of such obstructions is a difficult problem in management.

Peritoneal fluid occasionally collects in the lesser omental sac above and slightly posterior to the stomach. The opening into the sac, the foramen of Winslow, sometimes is sealed by exudative inflammatory changes

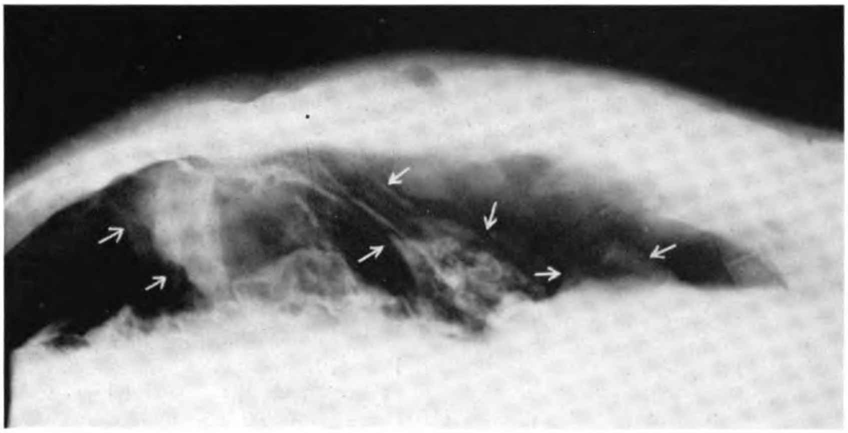

FIG. 161.—Artificial pneumoperitoneum reveals massive intra-abdominal adhesions. Transabdominal view shows sheets and strands of adhesions extending to anterior abdominal wall (arrows).

in adjacent organs. If a sufficient quantity of fluid accumulates, a cyst-like mass is produced which displaces the stomach and transverse colon downwards and to the left, producing a smooth, concave pressure defect. Perforation of the stomach or the biliary tract also is known to cause collection of gas and fluid in this space. In 1 of our cases the fluid collected as the result of congestive heart failure.

The radiologic identification of a strangulated loop of small intestine is important. In most cases a closed loop type of obstruction is present. The roentgen findings were discussed recently by Mellins and Rigler, who reviewed 26 cases. They found that in 60 per cent of their patients a closed loop was produced. If the twisted loop is partly closed, it contains gas and fluid, and on horizontal films the two limbs are separated by edematous intestinal walls, which they refer to as the "coffee-bean" sign. If the loop is completely closed, with little or no gas present, the loop presents as a rounded soft tissue density simulating a mass. Fixation of the loop in various positions and loss of normal mucosal patterns is observed. Another helpful sign is failure of the localized loop to be decompressed with nasogastric intubation. The involved segment in some is dilated out of proportion with the other intestinal loops. Rack and Glazer described a localized hazy shadow, sometimes with scalloped edges, attributed to fluid filled superimposed loops of decompensated lesser bowel. Hunt differentiated between strangulation and intact blood supply on the grounds that with a normal blood supply and simple obstruction the bowel forms a transverse ladder pattern with visible valvulæ conniventes, while strangulation causes distended loops with an irregular pattern and absent valvulæ conniventes.

A diagnostic method not utilized as much as it might be is artificial pneumoperitoneum for the demonstration of peritoneal adhesions. Perry, Von Drasher and Wangensteen recently reported that strangulated loops could be so identified as sausage-shaped densely homogeneous masses contrasting sharply with the normal loops.

In considering the cause of lesser bowel obstruction one must recall that large bowel obstruction can produce marked lesser bowel dilatation. If there is any chance of this, a barium enema examination is essential. The hazards of treating lesser bowel distentions due to colonic obstructions with nasogastric intubation was stressed by Michel, Thompson, Reinstine, Sentner and Dale.

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INTRA-ABDOMINAL ABSCESSES

Intra-abdominal abscesses often follow localization of an intraperitoneal inflammation, and may reach capacities of more than a liter. They appear radiologically like masses or cysts, the diagnosis sometimes being suggested by the presence of localized gas accumulations. Sometimes these re-

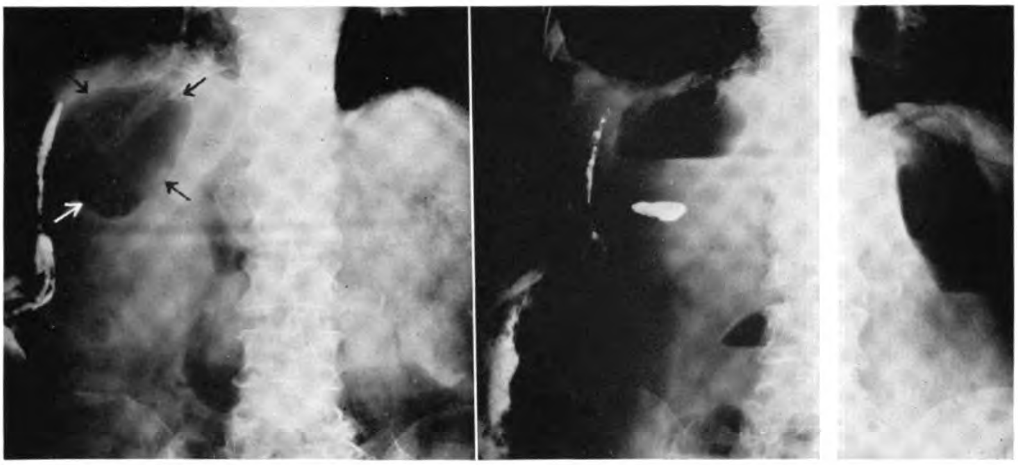

FIG. 162.—*A*, Postoperative subphrenic abscess in a 57-year-old woman who had been operated upon for a carcinoma of the cecum. A postoperative fistula formed to the right flank after abscess was drained. Lipiodol injection outlines the tract. A large gas containing cavity is present (arrows).
B, Erect film after Lipiodol entered the abscess cavity.

semble gas-filled intestinal loops, requiring contrast examination for accurate differentiation. Similar abscesses form retroperitoneally, particularly with intestinal perforation. In these the gas vesicles follow the fascial planes, outlining the kidneys, adrenals and psoas muscles.

Subhepatic purulent collections often are associated with perforations of the gallbladder or the adjacent colon, or a high gangrenous appendix. The demonstration of a pathologic gallbladder points towards a possible association, but barium enema examination should also be made to exclude a colonic point of origin. These masses sometimes become large enough to impair the movement of the right diaphragm, and can extend well into the liver substance.

Intrahepatic abscesses usually result from spread of pyogenic infection from the bowel through the portal veins. As a rule, these abscesses are multiple and communicate with one another. Sometimes they are sterile, and in many cases the cause cannot be ascertained even at operation or postmortem examination. A suggestion has been made that some minor lesion of the intestinal mucosa permits passage of bacteria into the portal veins. Unless these abscesses attain sufficient size, they cannot be identified radiologically. If breakdown of tissue occurs with gas formation, cavitation sometimes is identifiable on erect roentgenograms by a gas and fluid level.

Cholangiolytic abscesses result from common bile duct obstruction, and appear as multiple excavations which roughly follow the distribution of the intrahepatic ducts. Such cavitations occasionally are identified on retrograde cholangiograms.

Pus may gather beneath the diaphragm in six potential spaces, three under the right and three under the left leaf. On the right side are two intraperitoneal and one retroperitoneal spaces separated by the falciform ligament and the liver. The right anterior intraperitoneal space is continuous with the peritoneal cavity, and is bounded by the falciform ligament medially. The anterior space under the left diaphragm is situated anterior to and above the stomach. The posterior space under the right diaphragm is retroperitoneal, and is bounded by the liver anteriorly, with the kidney and duodenum forming its posterior and medial borders. The posterior space under the left diaphragm represents an upward extension of the lesser peritoneal sac. It is evident that the sub-

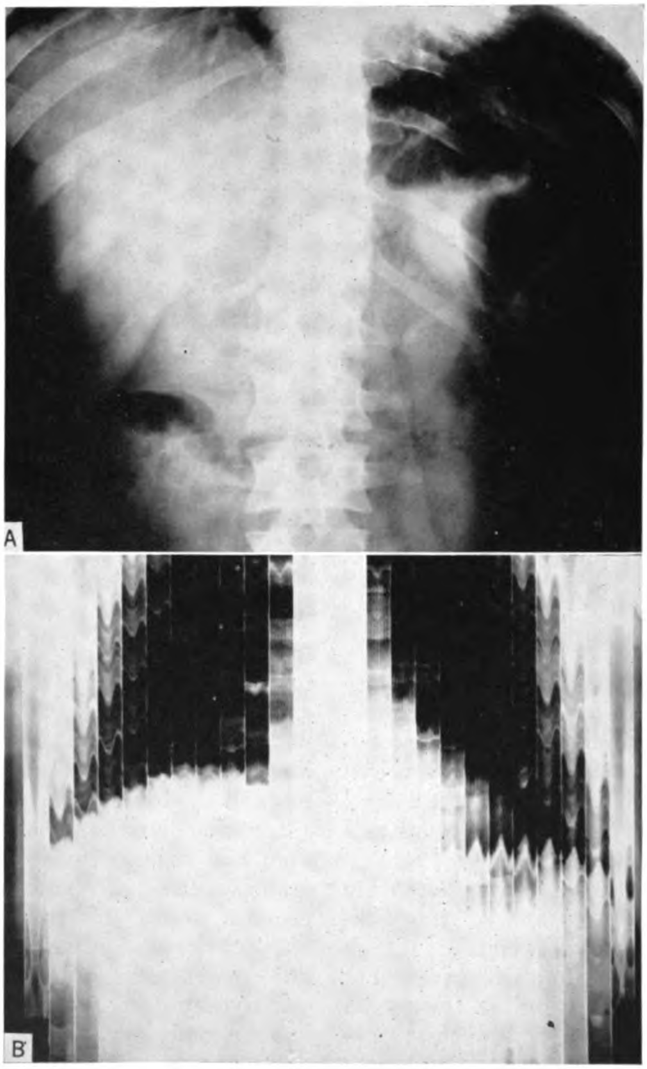

FIG. 163.—*A*, Right subphrenic abscess in a 30-year-old man after cholecystectomy. The right diaphragm is elevated.

B, Roentgenkymogram shows restriction of motion of the elevated right diaphragm.

phrenic space is a large one, and in general is defined by the diaphragm above and the transverse colon and mesocolon below. The subdivisions are not closely compartmented, and often are rather ill defined.

Right subphrenic abscesses follow extension of an inflammatory process, frequently acute appendicitis or pelvic abscess. These tend to localize in the anterior compartment. The posterior right subphrenic space becomes infected by extension from one of the retroperitoneal organs such as the duodenum, kidney, pancreas, lung or spine. Left anterior subphrenic abscesses may follow posterior wall gastric ulcers, pancreatitis, lesions of the left colon or retroperitoneal inflammations. Occasionally fluid due to peritonitis accumulates in the subdiaphragmatic spaces. Gallbladder disease, particularly when associated with cholangitis and pancreatitis, may produce intrahepatic abscesses with subphrenic or subhepatic components. Exten-

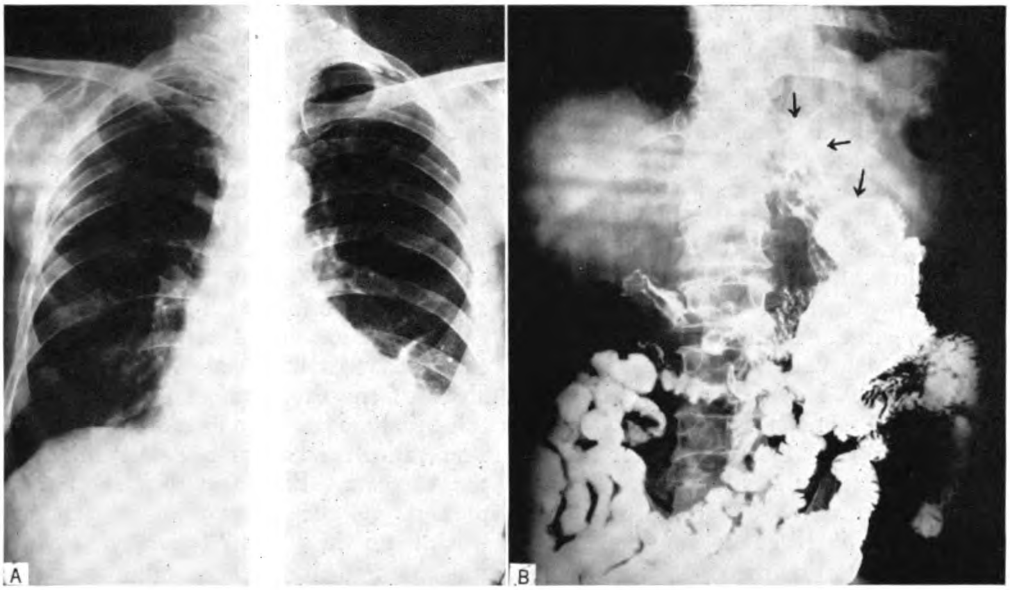

FIG. 164.—*A*, Chest roentgenogram of a 47-year-old man with a left subphrenic abscess following subtotal gastrectomy for duodenal ulcer. A pleuritic reaction obliterates the left diaphragm. The motion of the diaphragm is restricted.

B, Upper gastrointestinal examination of same patient. The upper aspect of the stomach is displaced downward and medially (arrows).

sion of subphrenic abscesses into the liver is not uncommon, and at times it is not possible to determine where the process originated.

No hard and fast rule is possible as to the distribution of subphrenic and subhepatic abscesses because of sites of origin of infection. It has not been rare to find a left subphrenic abscess associated with distant inflammation, sometimes on the right side. The contrary also has been observed.

With present methods of medical treatment, intra-abdominal infections often are treated successfully. In these cases the clinical picture of a subphrenic infection may be observed, but the radiological changes lag behind, so that an exact correlation cannot be made. The radiologic manifestions of subphrenic and subhepatic abscesses depend much on the bulk of the inflammatory lesion, as well as on the effects of inflammation on the serosal surfaces of the liver and diaphragm. If no fluid accumulates, the radiological changes may be minimal even if considerable inflammation exists. If these cases are treated successfully, the classical radiological pattern of subphrenic abscesses is aborted.

Under ordinary circumstances a subphrenic abscess may form within 1 to 8 weeks after an abdominal operation. Not infrequently a considerable delay ensues between these events, and periods up to a year or more may elapse before a subphrenic abscess is diagnosed. Most patients present symptoms of infection and pain which are rather inconstant and variable, and may reflect the secondary intrathoracic extension of infection or imitate other abdominal disorders.

A selected group of 50 subphrenic abscesses was reviewed. There were 25 on the right side, 22 on the left, and 2 were bilateral. One was situated under the middle leaf of the diaphragm. Of those on the right side, 10 followed gallbladder or biliary tract operations. Five followed surgery for acute gangrenous appendicitis, in 3 of which appendiceal abscesses were present. Two had perforating cecal carcinomas, and 2 others had perforating carcinomas of the ascending colon. In 1 patient pelvic peritonitis due to chronic salpingitis was found. In 1, a 70-year-old man, a prostatic abscess was considered the site of origin. In 1, the infected site was an ileal leiomyosarcoma found unexpectedly at necropsy.

The conditions associated with the left subphrenic abscesses were more diverse. Perforating carcinomas of the splenic flexure or descending colon were found in 8 patients. Perforated peptic ulcers of the stomach was contributory in 6. There were 3 with acute hemorrhagic pancreatitis, in 2 of which the condition followed operations for duodenal ulcer. In 1, pancreatitis followed blunt abdominal trauma. Peritonitis after appendectomy was seen in 1, and after cholecystectomy in another. A large seropurulent collection was observed in a patient after thoracolumbar sympathectomy for hypertension. In 1 patient a perforating benign granuloma of the splenic flexure was present. There were 2 patients who had had appendectomies from 2 to 8 weeks before the onset of abdominal symptoms. In 1, the clinical diagnosis was a right subphrenic abscess, but at operation this was not found. At necropsy a left subphrenic abscess was disclosed. This had been suspected radiologically because of left pleural effusion, displacement of the splenic flexure as estimated by the position of the gas-filled bowel, and some impairment of diaphragmatic motion. In the second case the diagnosis was established and confirmed at operation.

One patient had a bilateral subphrenic abscess. He had had a perforated gangrenous appendix with an abscess in the right subphrenic and intrahepatic area, identified at operation. Later he developed a fecal fistula from the splenic flexure, and a left subphrenic abscess appeared within 6 weeks after the first operation. At necropsy an unsuspected carcinoma of the jejunum was found. One patient had an abscess localized beneath the central tendon of the diaphragm after subtotal gastrectomy for a duodenal ulcer. He developed an external fistula, and later a collection of gas was noted immediately beneath the middle leaf. No impairment of motion of either the right or left diaphragm was present. A localized collection of pus was found in the anterior aspect of the epigastrium extending to the left upper quadrant. Apparently this abscess had formed at the site of a drain placed in the fistulous tract.

The radiologic changes produced by the right subphrenic abscesses in this series included an appreciable increase in the area of liver density in 12, associated with a somewhat indistinct delineation of the liver edge. Gas shadows within the subphrenic area were seen in 6 with large collections of gas and fluid. In 4 others only a small collection was observed. If a subphrenic abscess forms shortly after operation, the gas collection may have to be differentiated from air retained within the abdomen incident to the surgery. Elevation of the right diaphragm more than two intercostal spaces was present in 8. In 7 others the elevation was about one interspace, and in the remaining 10 the degree of elevation was insignificant. A considerable right basilar pleural reaction was encountered in 12 cases, a slight or moderate effusion was seen in 7, and in the remaining 6 no demonstrable pleural fluid or parenchymal infiltration was observed. Pleural changes appeared in intervals from 10 days to 5 weeks after the presumable onset of the abscess. Following healing, the pleural space resumed a normal appearance over a period of 2 to 3 weeks.

The motion of the right diaphragm was studied fluoroscopically and by means of roentgenkymograms. The latter was preferred, because it provided a permanent record of diaphragmatic motion. Pronounced restriction of motion was seen in 15 patients. In 5 others a lesser restriction was present, and in the remaining 5 the movement was normal. While at operation the collection of fluid was mainly in the anterior compartments, it was often difficult to state whether the process was limited to one or another of the anatomic delineations. At necropsy associated abscesses in the liver parenchyma were found in 3 patients. In 4 others there was extension of the purulent process through the capsule of Glisson, into the liver immediately beneath.

Not included in this survey were cases in which the clinical diagnosis of subphrenic abscess was suspected after operation, but who had normal radiographic examinations. These patients had been on intensive antibiotic treatment, and the clinical responses suggested that infectious processes had subsided without progressing to points where they could have produced radiological changes.

The radiographic changes due to left subphrenic abscesses reflected the anatomic alterations incident to collections of fluid, mainly in the anterior compartment. In these cases downward displacement of the cardia and reflection of the stomach anteriorly was seen, together with displacement of the splenic flexure downwards and anteriorly. Plain abdominal films disclosed increase in the density of the left hypochondrium. The identification of gas collections was difficult because of the confusing intestinal shadows. Visualization of the cardia of the stomach with barium or air was found helpful in disclosing its position in relation to the diaphragm. Elevation of the left diaphragm was less common than that of the right, only a moderate upward displacement being observed in 10 cases. In 5 others a slight elevation was present, and in the others the left diaphragm was in its normal position. The incidence of pleuropulmonary changes was about the same as on the right side. The differentiation between a left subphrenic inflammation with a concomitant pleural reaction from changes incident to surgical manipulation was difficult in some cases. Restriction of motion of the left diaphragm was observed in only 7 cases.

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INFLAMMATORY DISEASES OF THE LIVER

Acute infectious hepatitis of viral origin occurs either from contaminated food, through contacts with carriers, or by exposure to infected blood. There may be an incubation period of up to 3 or 4 months before jaundice appears. The diagnosis in the pre-icteric stage is difficult, and laboratory examinations in this stage are not helpful.

In the icteric stage oral cholecystography is occasionally helpful when normal visualization is observed. As early as 1930 Rudisill noted good visualization in 7 out of 8 patients with catarrhal jaundice, and in 1933 Foley noted normal cholecystograms in 68 per cent of his cases. More recently, Cohn, Berger and Kremens reviewed cholecystography in 50 patients with jaundice due to varying causes such as acute hepatitis, cirrhosis, liver metastases, obstruction and hemolytic disease. The gallbladder visualized in 21 patients, all with a serum bilirubin level of 5.0 milligrams per cent or less. Opacification of the gallbladder with higher serum bilirubin levels was poor. This was equally true with infectious hepatitis as well as with extrahepatic obstructive jaundice. The presence of a normally visualized gallbladder increases the probability of jaundice of intrahepatic origin. Nonvisualization contributes little to differential diagnosis.

The normal level of bromsulfalein retention is about 20 to 23 per cent. With greater retention the gallbladder does not visualize. Nonvisualization with bromsulfalein retention of less than 20 per cent is suggestive of gallbladder disease as opposed to intrinsic liver disease according to Etess and Strauss. Jarviner and Paltia regarded bromsulfalein retention levels of 30 to 40 per cent as more critical, observing that with retention of more than 40 per cent cholecystography was unsuccessful, between 30 and 40 per cent uncertain, and under that concentration good visualization is obtained. These authors also noted that cholecystography in many cases of infectious hepatitis was normal. Among 23 such cases they reported that in only one was nonopacification obtained, and this patient had gallstones.

Serial oral cholecystographies with increased doses of contrast medium have been advised in doubtful cases. In our experience it has been uncommon for the gallbladder to fill under conditions in which it failed to do so using the customary 3 gram dose of Telepake for patients of average size. We have repeatedly shown that when infection subsides a similar test dose 1 or 2 weeks after an initial non-visualization results in good opacification, while repetition with a double or triple dose in a day or two disclosed no opacification unless the patient's condition had improved markedly.

The pathologic change due to viral hepatitis is a cholangitis and cholangiolitis, the prime lesion being damage to the parenchymal cells. This varies from swelling of the cells with disintegration of the nucleus to massive central necrosis of the lobules. Swelling of Kuppffer's cells, edema and cellular infiltration of the portal and periportal areas also are present.

Varying degrees of hepatomegaly are encountered, and occasionally the spleen also is enlarged. Right upper quadrant pain appears relatively late, and symptoms may abate when jaundice appears. The degree of visualization of the gallbladder parallels to some extent the liver changes as jaundice deepens and recedes later. Intravenous cholangiography also parallels the pathologic changes in the liver. In the absence of advanced hepatic disease good filling of the biliary system may be anticipated when the bromsulfalein and serum bilirubin changes indicate little hepatic damage.

The most common inflammatory disease of the liver is cirrhosis. In most instances its cause is unknown. Coexistent alcoholism is rather frequent, but no absolutely definite etiologic relationship has been established. Cirrhosis also is seen with other diseases such as hyperthyroidism and ulcerative colitis, and sometimes follows chemical poisoning with agents such as carbon tetrachloride and phosphorus. The pathologic changes of portal cirrhosis include dense perilobular connective tissue infiltration, degeneration of liver cells, dissociation of liver cords combined with degeneration and fatty and cellular infiltration. Necrosis of liver cells is believed to precede perilobular fibrosis. Later the liver becomes irregularly nodular, hard and shrunken. Associated with this are changes produced by portal venous obstruction as manifested by ascites, venous congestion, varices and congestion and edema of the intestinal walls. The liver sometimes enlarges in the early stages of the disease, but it is far more common in later stages to find a shrunken liver weighing less than 1500 grams.

The early clinical diagnosis of cirrhosis is difficult, the disease often being unnoticed because of paucity of complaints. With advanced liver damage, evidences of portal hypertension such as ascites and gastrointestinal bleeding appear. Jaundice comes on gradually and disappears, only to recur in a more intense form later. The association of biliary duct or hepatic carcinoma with cirrhosis must be kept in mind. Parker reported carcinomatous changes in 30 or 184 patients with cirrhosis who came to necropsy, a figure about 4 times that in the American literature.

Radiological investigations in patients suspected of cirrhosis are non-contributory in the early stages. Oral cholecystography usually is normal, but with the onset of parenchymal damage the extent of opacification diminishes. Cuniff, Dolan and Leevy reviewed 50 cases of portal cirrhosis, and noted that repetition of oral cholecystography with double doses in 9 patients who had absent visualization with single doses, revealed better opacification in only one.

The radiologic demonstration of esophagogastric varices is important in the investigation of upper gastrointestinal bleeding.

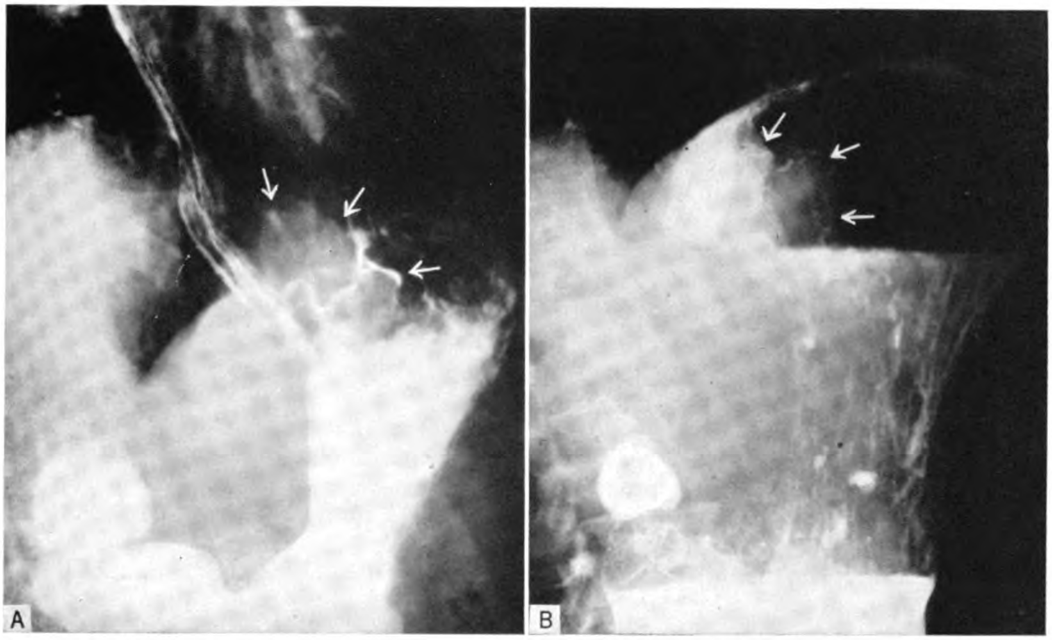

FIG. 165.—*A*, Esophageal varices in a 56-year-old patient with cirrhosis of the liver. The esophageal mucosal folds are widened. A mass is present in the cardia (arrows).

B, Erect stomach roentgenogram, same patient. The mass in the gastric cardia (arrows) was found to be due to extrinsic pressure from an enlarged quadrate lobe of the liver.

The diagnosis depends on the identification of dilated veins protruding between the normal linear esophageal folds. Dilated veins often extend well above the lower esophagus, and may protrude into the stomach as well. Occasionally they are large enough to cause esophageal obstruction or to simulate carcinoma of the stomach. We have seen 5 cases in which the diagnosis of carcinoma of the cardia was made because of irregularly lobulated masses protruding into the stomach gas bubble. Bleeding was but a minor manifestation in 3 of these patients.

Cirrhosis produces mucosal changes in the stomach and lesser bowel, sometimes associated with considerable ileus. Ascites is suggested by a diffuse, hazy opacity of the abdomen, and is frequently associated with moderate ileus. Examination of the gastrointestinal tract in these patients discloses slow passage of barium through the intestinal tract, often associated with irregular distention and flattening of the mucosal folds. A more acute mucosal change was described by Palmer, who reported erosive gastritis attributed to the influence of portal hypertension on the gastric mucosa. While uncommon, this may cause profuse bleeding. Palmer found this to be similar to nonspecific gastritis both gastroscopically and histopathologically.

Plain abdominal films often disclose moderate to fairly well-advanced splenomegaly with a small liver. Occasionally intrahepatic areas of radiolucent foci are identified, attributed by Steinbach, Crane and Bruyn to fatty alteration within the parenchyma.

Hepatic cirrhosis in children is relatively uncommon. We saw such a case recently in a 6-week-old child with ascites. On paracentesis purulent fluid was obtained, and because a peritoneal infection was postulated, laparotomy was performed. The mesocolon and mesentery were markedly edematous, and the spleen was slightly enlarged. The liver was somewhat firm, but its surface markings and color were normal. No points of perforation of the intestines could be found. The patient expired about a week later, and necropsy revealed a characteristic picture of cirrhosis, with focal hemorrhages in the small intestine and lungs.

Occasionally cirrhosis causes atypical configurations of the liver, particularly its left lobe. In 1 case the left lobe was enlarged, and impinged on the cardia. The resulting defect was attributed to carcinoma of the cardia. The patient had slight bleeding from the upper gastrointestinal tract, and small esophageal varices were present. Since the possibility of the defect in the stomach being due to varices was discounted, operation was advised. At laparotomy the gastric defect was found to be due to extrinsic pressure from a nodular, hypertrophied left lobe of the liver. (Fig. 165).

With the advent of shunting operations for relief of portal hypertension, visualization of the portal circulation is important. Such examinations are performed by injection of contrast materials directly into the spleen percutaneously, by injection of the portal vein at operation, or by transhepatic injection. These examinations are best done under rigid control, with the operating room prepared. They should not be attemped unless a shunting operation is contemplated. Visualization of the hepatic circulation has been achieved by aortography at the level of the eleventh thoracic vertebra. The resultant spleno-porto-hepatograms reveal diminished circulation in cirrhotic livers, and are important in evaluating the circulation of the portal system and of the perigastric and periesophageal venous trunks. The information derived from visualization of the hepatic venous or arterial bed requires further evaluation.

Cirrhosis of the liver also is produced by chronic inflammation of the biliary radicles. In its primary form, biliary cirrhosis is relatively uncommon. It occurs more often in women, and is characterized by jaundice and considerable hepatosplenomegaly. The liver remains smooth, and the smaller hepatic radicles show evidences of cholangitis and pericholangitis. Involvement of the liver cells does not occur until late. The disease pursues a slow, progressive course. As jaundice develops, the disease simulates portal cirrhosis. A differential diagnosis often is quite difficult, and radiologic methods of investigation are not particularly helpful.

Cirrhosis also results from biliary tract obstruction and inflammation. Occasionally this is produced by congenital defects such as atresia of the common bile duct or a major duct. Parasitic infestations of the liver are another etiologic agent. Such biliary cirrhosis may terminate fatally unless the cause of obstruction is removed. In these patients percutaneous, transhepatic or operative intrahepatic cholangiography is a most useful procedure for identifying the ductal system.

An interesting form of veno-occlusive liver disease in West Indian children and adults was described by Stuart and Bras. The primary lesion in the acute stage is widespread occlusion of the smaller and medium sized hepatic veins and sinusoidal congestion with pressure necrosis of the periportal parenchyma. Hepatomegaly and ascites appear as a result of obstruction of the hepatic venous outflow. This disease either subsides or progresses to cirrhosis with ascites, hepatosplenomegaly and infrequently, jaundice. These authors demonstrated roentgenograms of liver specimens injected with barium which disclosed that only the main hepatic vessels filled, while the smaller vessels were not opacified.

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INFLAMMATORY DISEASES OF THE BILIARY TRACT

In view of the progress being made in cholecysto-cholangiography a note as to technic of examination for inflammations of the biliary tract is in order. The examination begins with scout films, which often disclose calcified gallstones or a dilated gallbladder. Occasionally gas in the biliary ducts indicate the possibility of a fistula or a previous surgical procedure involving the biliary tract. The basic diagnostic procedure is oral cholecystography. Three grams of Telepake is an acceptable routine for patients weighing less than 180 pounds. Heavier individuals are given from 4 to 5 grams, but this is not essential. Films are made 12 hours later in various prone and erect projections, and occasionally spot films under fluoroscopic control are obtained with the patient erect. Reexaminations of non-visualized gallbladders or the preliminary use of double doses are no more conclusive than a well-performed routine examination. Occasionally, examination with double and triple doses will permit some opacification of a previously non-visualized gallbladder, thereby permitting the identification of radiolucent stones. Non-visualization is indicative of impaired absorptive function of the gallbladder mucosa. If operation is contemplated, and conditions permit, we usually check with a repeat study 1 to 2 weeks later using the same dose of contrast material. Not infrequently, particularly after a bout of acute cholecystitis, the function of the gallbladder returns and reveals stones. The presence of a normal visualization soon after absent opacification brings up the question as to whether a non-calculous cholecystitis had been present. It should be recalled that other causes of absent visualization exist, such as recent severe abdominal trauma or renal colic.

It is helpful to administer a fatty meal just before taking the 12-hour films. This often results in better identification of the cystic and common ducts. Pharmacological aids in visualization of the biliary system also may be utilized. Paregoric and other opium derivatives produce spasm of the sphincter of Oddi, improving the opacification of the biliary ducts. The combination of paregoric with heavier doses of contrast medium for opacifying the common bile duct after cholecystectomy was recently proposed by Lowman and Lawson.

In evaluating oral cholecystography, one must be sure that the proper dose had been taken, and that the contrast medium entered the intestinal tract. If the test dose is retained in the stomach, the examination obviously is unreliable. This may occur because of pyloroduodenal obstruction incident to duodenal ulcer or the use of drugs which inhibit gastric motility. Other causes include disturbances of intestinal absorption or rapid transit through the intestinal tract, although these are rather infrequent in my experience. If contrast medium is noted in the stomach, a film made after 36 hours will

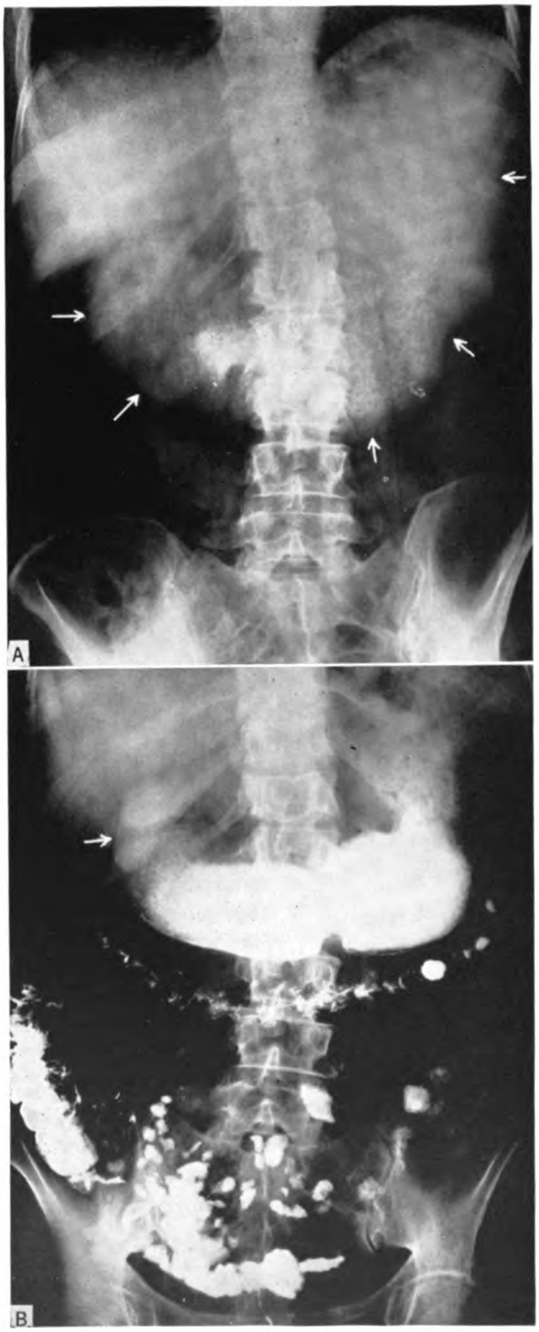

FIG. 166.—*A*, Abdominal film 12 hours after intake of 3 grams of Telepaque. The stomach is distended with fluid (arrows), and the contrast material has not passed into the lesser bowel.

B, Same patient, 36-hour film, 24 hours after a barium meal. Note the gastric retention. Apparently the Telepaque has entered the bowel and been absorbed, inasmuch as the gallbladder is visualized (arrows). Patient had a cicatrizing duodenal ulcer.

not infrequently disclose a normal functioning gallbladder. Recently we had 1 patient in whom the intact pills passed through the intestinal canal. He had a normal visualization after being given the same dose in powder form.

Intravenous cholangiography with Cholegafin is useful for visualizing the biliary system in patients who have non-functioning gallbladders as demonstrated by oral cholecystography. It does not replace the conventional examination because it is not a test of the function of the gallbladder mucosa. However, it is of considerable importance in the examination of post-cholecystectomy patients for demonstration of the common bile duct. Failure to enter the biliary sac is presumptive evidence of cystic duct obstruction. Occasionally the distal portion of the common duct does not visualize during intravenous cholangiography, so that stones in this are undiagnosed. Visualization of the ductal system is not constant even in patients in whom we ordinarily anticipate a satisfactory result, and for no apparent reason. Oral cholecystography followed immediately by intravenous cholangiography sometimes is a superior method for outlining the gallbladder and its passages. Intravenous cholangiography is usually unsuccessful in patients with jaundice when the serum bilirubin level is over 5 mgm. per cent, or when the bromsulfalein clearance is between 20 and 30 per cent. We have not had perceptibly better results when carrying these examinations to 24 hours.

Percutaneous transabdominal cholangiography is best done in patients with obstructive jaundice to localize obstructive sites. Operative cholangiography also enables obstructions to be identified, particularly at the distal end of the common ducts or within the liver substance. Postoperative "T" tube cholangiograms serve to provide similar information.

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Cholecystitis in Children.—Acute cholecystitis is more frequent than chronic cholecystitis. Jaundice is fairly frequent because the gallbladder and common duct are rather close, permitting intermittent obstruction. Cholelithiasis is infrequent. In a series of

30 cases of proved childhood cholecystitis Ulin, Nosal and Martin found stones in 57 per cent, and in 7 per cent stones were present in the common duct also. About half of these patients were jaundiced. Cholelithiasis has been related to excessive excretion of pigment due to hemolytic anemia. Griffin and Smith found gallbladder disease a complication in more than half their patients with hemolytic anemia.

The radiologic investigation of the gallbladder in children follows the same procedure as in adults. We use 1.5 grams of Telepake in children who weigh 50 pounds or less. In children who do not swallow the tablets it is best to administer crushed tablets through a nasogastric tube, making sure to flush the tube thoroughly before withdrawing it. Intravenous cholangiography has relatively little application. However, operative cholangiography is a diagnostic necessity in the radiologic exploration of the biliary tree for obstructions.

Acute and Chronic Cholecystitis.—This occurs most often in women in the fourth decade of life. Symptoms often are sudden in onset, but previous distress and digestive disturbances are frequent. Acute episodes are characterized by epigastric pain which settles in the right upper quadrant. Scout films at this time may disclose a faintly outlined globular shadow which represents a swollen, tense gallbladder. Oral cholecystography usually disclosed non-visualization. When the acute phase subsides, repeat examination often reveals a return of function, sometimes within a week or 10 days. The absence of gallstones serves to identify the disease as non-calculous cholecystitis. Nevertheless, in several such cases observed by us small pigment stones were present at later operation even though careful oral cholecystography was normal. Often small stones are best identified on films showing a contracted, almost empty gallbladder.

Chronic non-calculous cholecystitis is associated with symptoms of biliary dysfunction. Oral cholecystography discloses persistent diminution of concentration or non-visualization, the former often being quite difficult to evaluate. Intravenous cholangiography is useful in this situation, because entrance of the contrast medium into the gallbladder serves to demonstrate patency of the cystic duct and the presence of stones.

Acute gangrenous cholecystitis can progress to perforation, with pericholecystic infection and abscess formation. A large granulomatous mass sometimes can be identified on scout films. If this reaches sufficient size it may deform the adjacent transverse colon or hepatic flexure by extrinsic pressure. Envelopment of the bowel by the inflammation can simulate intrinsic neoplastic disease. As a rule, oral cholecystography shows nothing more than failure of visualization and intravenous cholangiography is not particularly helpful. If gangrene causes intraperitoneal perforation with expulsion of bile an acute and often fatal bile peritonitis results.

Acute emphysematous cholecystitis, or pneumocholecystitis, results from infection with gas-forming organisms such as Cl. perfringens or E. Coli. This condition cannot be differentiated clinically from acute cholecystitis. Radiologically, the presence of gas within the walls of the gallbladder produces a picture which is quite diagnostic because of the radiolucent band which defines its periphery. When the sac itself is filled with gas, pneumocholecystitis ensues, and the radiolucent sac may be identified on plain films. The gas also can enter the biliary ducts if the cystic duct is patent. Most of these patients have gallstones, and it is believed that the condition is initiated by cystic duct obstruction followed by decreased local resistance of the gallbladder wall, increased activity of the bacterial invaders and production of gas in the gallbladder wall extending into the biliary sac. Other mechanical obstructions such as strictures, kinks, anomalous vessels and adhesions can set up a similar mechanism. The possibility that pericholecystic gas is due to penetration into the Aschoff-Rokitansky sinuses was suggested by Rocco, Hunt and Sadran.

Cholecystitis and Cholelithiasis.—This is by far the most frequent gallbladder disease. In addition to the inflammatory changes in the wall, intraluminal stones may vary from tiny concretions to large, facetted calculi. Radiotransparent stones are composed mainly of cholesterol and bile pigment, while the opaque variety contains sufficient calcium to

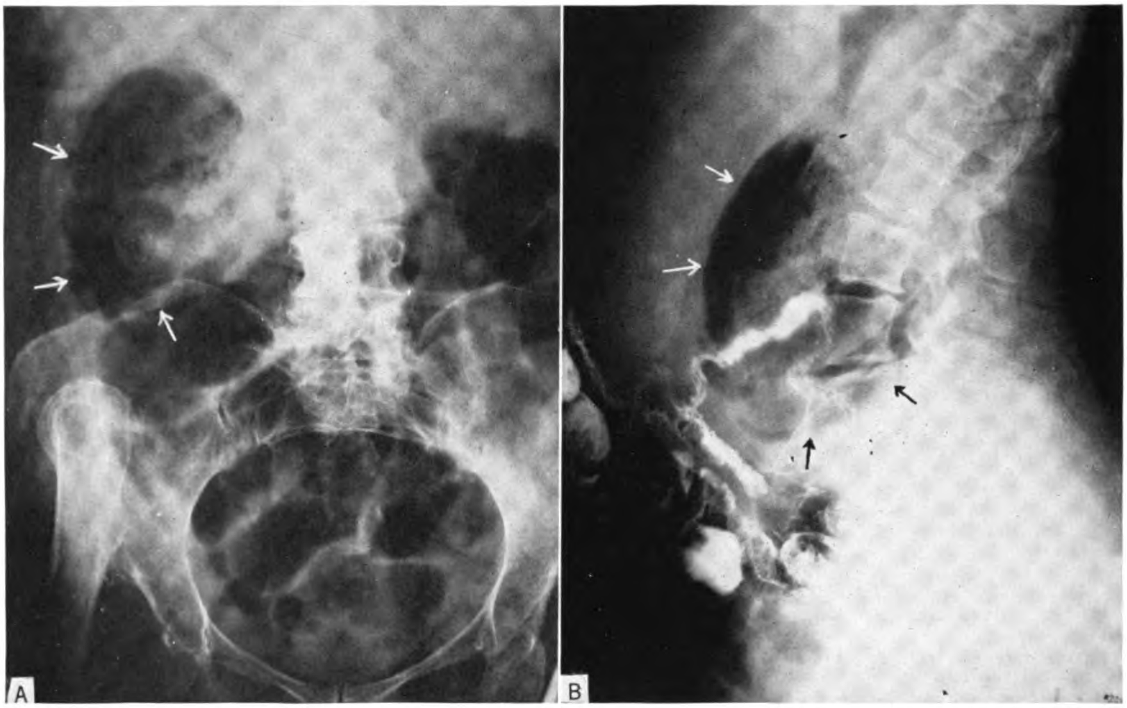

FIG. 167.—*A*, Pericholecystic abscess in a 72-year-old woman with a palpable mass in the right upper quadrant. The gas in the abscess cavity surrounds the enlarged gallbladder (arrows) At operation a perforation was found in the posterior aspect of the gallbladder adjacent to the cystic duct. The large abscess cavity extended posteriorly.

B, Same patient, lateral view, showing the posterior extent of the abscess (arrows).

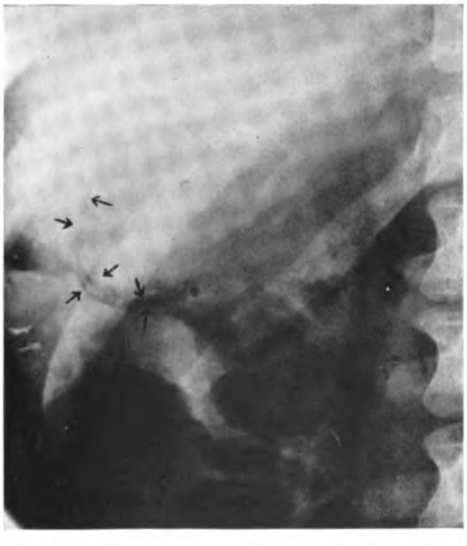

FIG. 168.—Gangrenous gallbladder with gas in its walls (arrows). Patient was a 61-year-old man with acute epigastric pain, chills, fever and jaundice. Verified at operation.

render them visible. An interesting form of cholelithiasis is that referred to as limy bile, or milk of calcium bile, which is a creamy precipitate containing small stones. This is best identified on erect examinations, which disclose a layering of this grumous material. The clinical picture is dependent on the effects of inflammation as well as the mechanical effects of passage of stones into the ducts. Silent gallstones are frequently encountered, and the question often arises as to whether or not such patients should be operated upon. In a review of 112 such cases Comfort, Gray and Wilson noted that approximately half developed symptoms. They found no relationship to the development of carcinoma, and regarded cholecystectomy as optional but did recommend operation if symptoms increased.

The radiologic examination should not stop with the demonstration of stones on scout films. Oral cholecystography is required

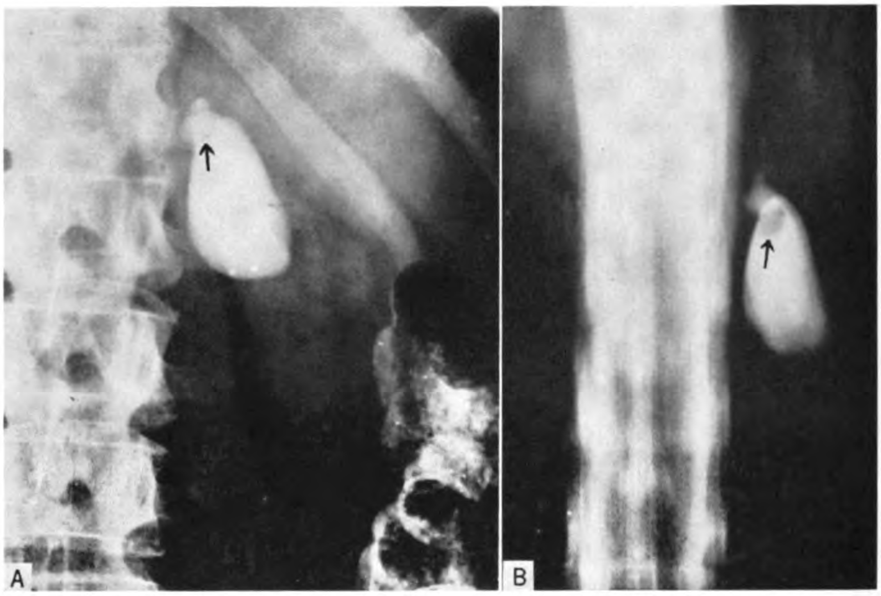

FIG. 169.—*A*, Oral cholecystogram shows normal visualization of the gallbladder. A faint shadow is present near the top of the sac (arrow).
B, Same patient, body section roentgenogram shows a definite stone (arrow).

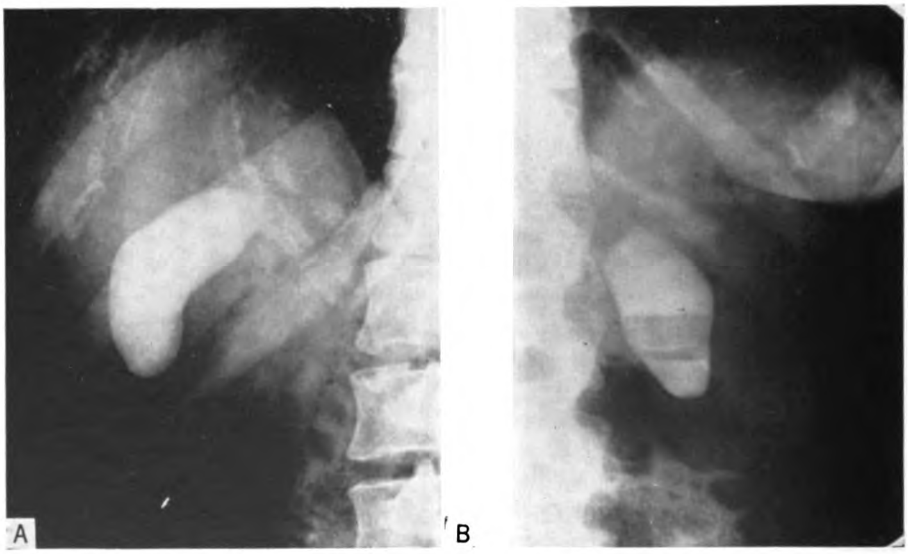

FIG. 170.—*A*, Intravenous cholangiogram in a 21-year-old girl who had nonvisualization with oral cholecystography. The sac is well opacified, and small stones are faintly seen in the fundus.
B, Same patient, erect roentgenogram shows a horizontal radiolucent band of "floating" gallstones.

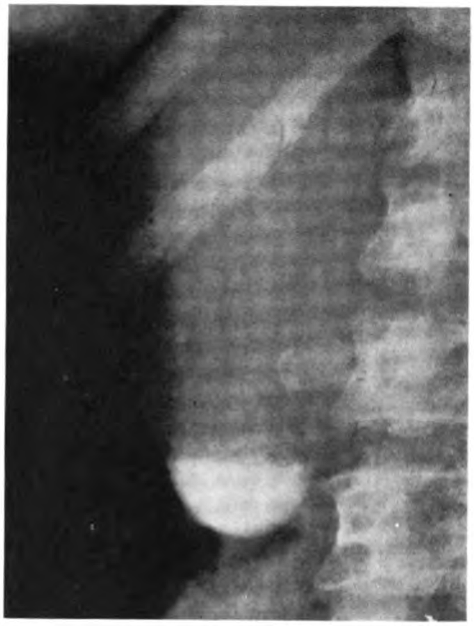

Fig. 171.—Erect scout film of abdomen in a 46-year-old woman with milk of calcium bile. Note the layering of the dense calcific material in the gallbladder.

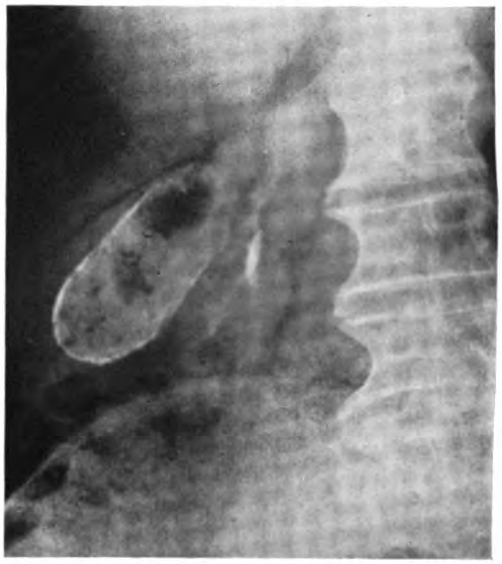

FIG. 172.—Calcified gallbladder in a 75-year-old woman. Asymptomatic at the time this film was made during intravenous urography.

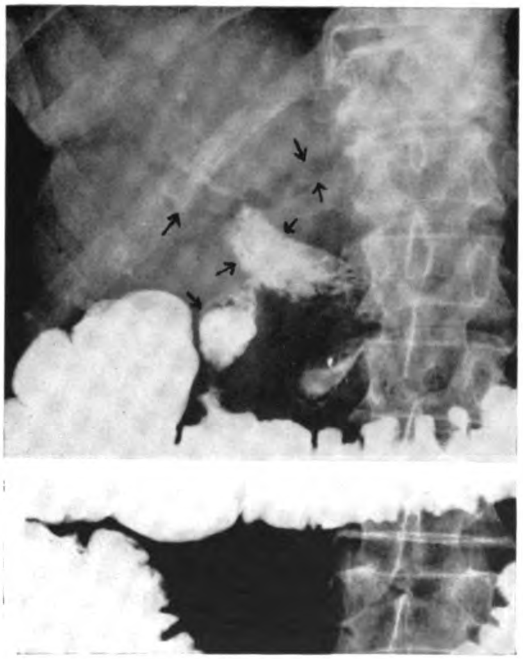

FIG. 173.—Cholo-cholecystic fistula in a 68-year-old woman with chronic cholecystitis (arrows). Barium and air are present in the ductal system.

for demonstrating the functional capacity of the biliary sac. The cystic and common ducts should be visualized as well. If oral cholecystography is unsuccessful, intravenous cholangiography may be required to pass on the patency of the cystic duct.

Hydrops of the gallbladder follows obstruction of the cystic duct by stone, tumor or inflammation. Sometimes the sac becomes large enough to be palpated, and it may show up on scout films with an almost cyst-like density.

The variety of sizes and shapes of gallstones is remarkable, varying from limy bile to stones more than 5 cm. in diameter, and their configurations may be facetted, round, smooth, irregular or mixed. Sometimes they resemble renal calculi. The internal structure also differs widely, from laminated stones to those with stellate gas inclusions. An interesting variety is the floating stone, seen on erect studies as a radiolucent band midway in the biliary sac. No specific clinical significance is attached to this variation. Non-visualization during and soon after an attack of biliary colic is frequent. A repeat examination should be done to determine return of function.

Cholecystitis is accompanied by reflex and inflammatory changes in the adjacent viscera

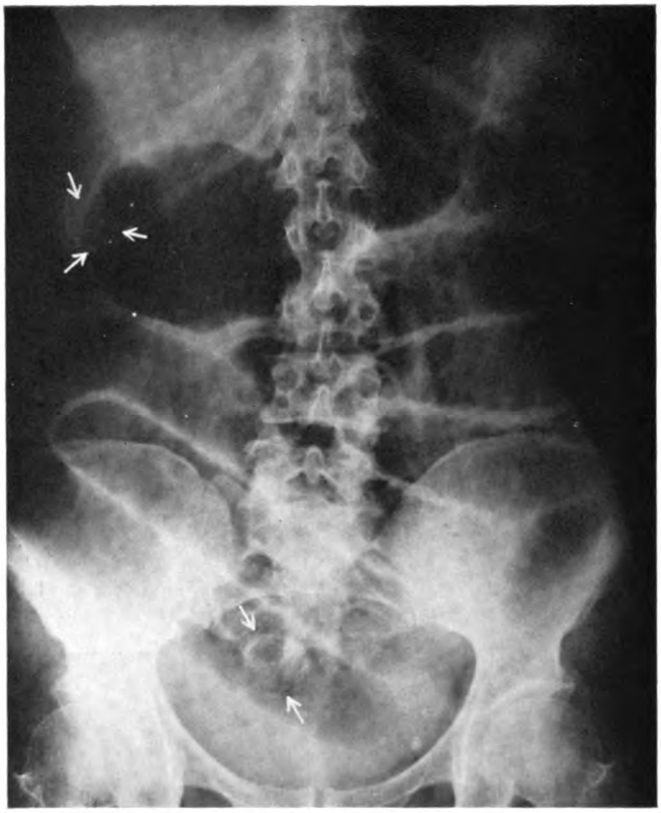

FIG. 174.—Gallstone ileus in a 65-year-old woman with a cholecystoduodenal fistula. Gas is seen in the biliary ducts (upper arrows). A stone is visible in the gas-filled ileum in the pelvis (lower arrows). Another stone is impacted in the distal ileum in the right upper quadrant (middle arrows).

rather frequently. This may produce no visible abnormalities. However, occasionally a deformity of the pyloroduodenal segment is seen which is indistinguishable from a peptic ulcer, or if extensive, an antral malignant process. One should not be overenthusiastic in attributing such deformities to biliary tract disorders.

Egg-shell calcification of the gallbladder wall is sometimes sufficiently dense to be identified on scout films. This usually follows cholecystitis with extensive damage of the wall. The clinical pattern of calcareous gallbladder disease is not different from that usually seen with preexistent acute cholecystitis.

Cholecystoduodenal and cholecystocolic fistulas are infrequent complications of cholecystitis. Rarely fistulous tracts to the stomach and retroperitoneal area are seen. Bronchobiliary and pleurobiliary fistulas as a rule follow trauma, but may be of infectious origin. If the pleural cavity is accessible, a pleurobiliary fistula forms, with bile accumulating in the thorax. This may become infected, forming an empyema. If the pleura is fused to the diaphragm, the fistulous tract enters the lung and bronchial tree. Adams reported 2 cases of bronchobiliary fistulas which were caused by common duct strictures. The complexity of these, and the difficulty of demonstrating them either by bronchography, cholangiography or at operation was striking. In a review of 819 cases of fistulas involving the gallbladder Waggoner and Le Mone found that 91 per cent involved the colon or duodenum. These follow cholelithiasis, acute cholecystitis, gangrene and pressure necrosis with perforation of the gallbladder. Gallstones entering the

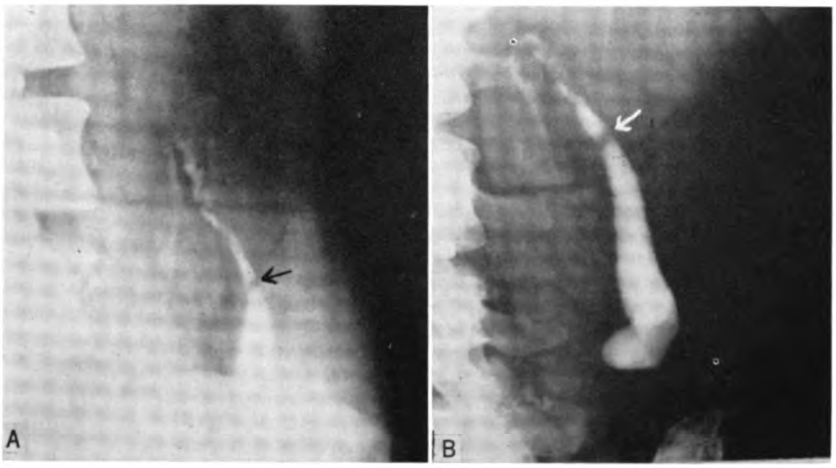

FIG. 175.—*A*, A small filling defect is present at the apex of the gallbladder in this 38-year-old woman (arrow).

B, Same patient 2 weeks later. The defect persists, and was diagnosed as localized cholesterosis. Operation not considered advisable at this time.

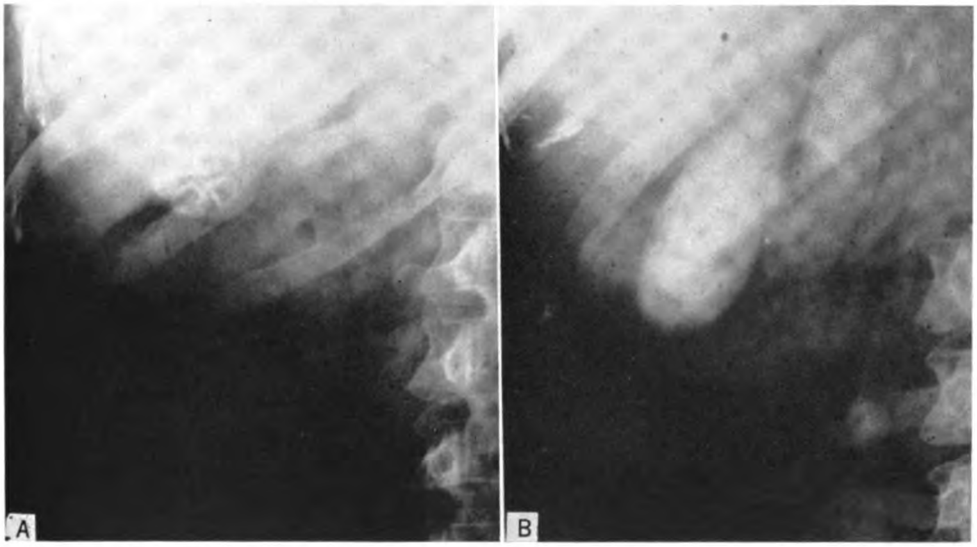

FIG. 176.—*A*, Oral cholecystography in a 55-year-old woman reveals multiple facetted calcified gallstones.

B, Same patient 8 years later. The gallstones have disappeared, and the gallbladder visualizes well. There had been no operation.

small intestine may produce obstruction. While small stones are readily evacuated per rectum, larger ones may obstruct the bowel at the duodenum, at the level of the ligament of Treitz, or at the ileocecal valve. These are the most frequent sites of blockage, but obstruction may occur anywhere in the small bowel. The possibility of multiple large stones should not be overlooked.

The radiologic picture of fistula formation is characterized by entrance of air into the biliary tree. A similar appearance may be due to previous operation such as sphincterotomy or surgery for relief of obstructive jaundice. Gallstone ileus follows the pattern of intrinsic obstruction, and the level of the impaction of a gallstone is sometimes determined accurately by identification of the stone itself. This is relatively easy if the stone contains calcium. With radiolucent stones, the point of obstruction is occasionally made out by demonstration of the convexity of the stone against the adjacent air in the bowel.

Cholesterosis.—Often called "strawberry gallbladder," this condition refers to mucosal deposition of yellowish-white cholesterol which shows up in striking contrast to the deep red mucosa beneath. Oral cholecystography is normal with the diffuse form of cholesterosis. The papillomatous form of cholesterosis may result in single or multiple small filling defects fixed to the wall of the gallbladder. Nonopaque gallstones sometimes are coexistent, but can be differentiated by their mobility. Papillomatous growths present similarly fixed shadows, but can be diagnosed with certainty only by histologic examination. A relationship between cholecystitis and cholelithiasis and cholesterosis often has been suggested. Such association was reported in about 11 per cent of cases by Feldman and Feldman, but others give figures as high as 50 per cent.

A mystery infrequently encountered in clinical practice is the spontaneous disappearance of gallstones. We saw this in a 55-year-old woman, who in 1940 had multiple gallstones with heavy calcific rims. Eight years later she returned for reexamination, and oral cholecystography showed a normal gallbladder without stones. There had been no operative intervention during this time (Fig. 176).

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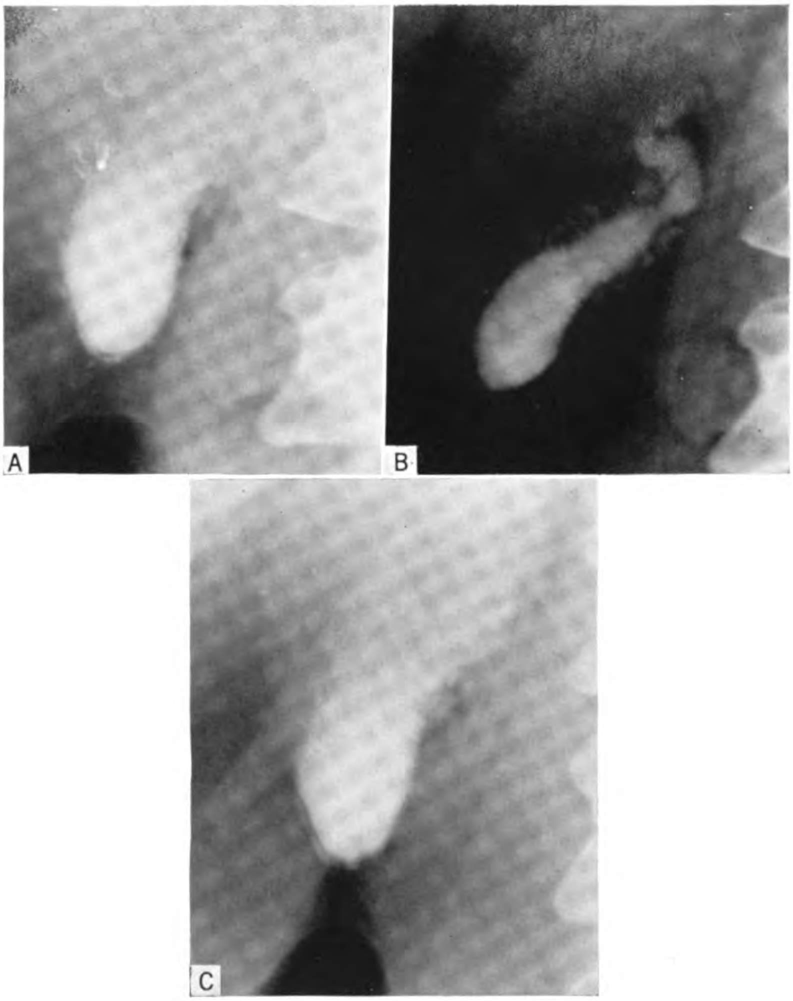

FIG. 177.—Various examples of Rokitansky-Aschoff sinuses. Note the irregularity and pouch-like deformity of the wall of the gallbladder. (Courtesy of the Armed Forces Institute of Pathology, section of Radiology, W. L. Thompson, Col., M.C. [ret.].)

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Rokitansky-Aschoff Sinuses.—In 1927 Halpert reported morphological studies of the gallbladder, outlining the pathologic changes associated with Rokitansky-Aschoff sinuses and Luschka's ducts. The latter are structures along the periphery of the gallbladder with histologic characteristics of intrahepatic bile ducts. The former represent projections of gallbladder mucosa into the muscular coats, the finger-like projections reaching the serosa. The tendency for Rokitansky-Aschoff sinuses to be accompanied by narrow irregular channels producing small cyst-like structures was brought out by Robertsson and Ferguson. These authors recognized the possibility of formation of nodular structures by localized collections of such cysts, which in turn produce nodular deformities of the gallbladder wall often referred to as adenomas. Le Quesne and Ranger reported a group of 12 cases in which such nodules produced filling defects in the fundus of the gallbladder, and suggested that cholecystitis glandularis proliferans constituted an entity, the specific feature being a localized area of penetrating epithelial sinus formation with muscular hypertrophy. This either causes no symptoms, or is associated with complaints suggestive of cholecystitis and cholelithiasis. Such nodular shadows cannot be distinguished radiologically from adenomas, but certain signs such as a funnel-like filling defect, sometimes with a central pit, dye-filled spaces around the periphery of the gallbladder and a cystic duct stricture are suggestive. All depend upon retention of concentrating power by the gallbladder. The tendency for these structures to show up best on films after a fatty meal was noted.

In a study of 495 gallbladders, Robertsson and Ferguson identified three groupings of Rokitansky-Aschoff sinuses. These included diverticula which reached the muscularis, those found in the intramuscular lacunæ, and those which penetrated the muscularis to reach the serosa. Normally the gallbladder contains few such structures, and these are quite superficial. With inflammation, and particularly with obstruction at the cystic duct increasing the pressure within the gallbladder, these sinuses become frequent, deeper and more developed. They also increase in proportion to age. Often bile pigment and cholesterol crystals, and sometimes stones are enclosed. Inflammatory changes usually are present. While these alterations are seen frequently on pathologic examination, their demonstration radiologically is relatively uncommon. This is due to the failure of visualization because of chronic inflammation. These sinuses appear as protrusions within the periphery of the visualized gallbladder, either as serrated or sometimes as irregularly linear transparencies separating the visualized portion of the gallbladder from the true wall. In some cases actually thickening of the wall of the gallbladder can be demonstrated. These structures are seen to better advantage on films made after a motor meal.

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Dysfunction of the Sphincter of Oddi.— The distal end of the common duct, including the sphincter of Oddi, is prone to intrinsic changes which cause either partial or complete extrahepatic biliary obstruction, with symptoms indistinguishable from gallbladder disease. In many, obstruction is insufficient to produce jaundice, and oral cholecystography is normal, although some dilatation of the common duct may be seen. Intravenous cholangiography is more suitable for demonstrating such dilatation or prolonged opacification indicating partial obstruction. It is difficult to set an absolute value for the size of the common duct. Between 7 and 10 mm. is approximately correct. The shape of the duct as seen on intravenous cholangiograms may be helpful, tapering of the distal end being suggestive of some pathologic change. In a considerable number of cases, the distal end of the duct does not opacify. Under such circumstances caution should be exercised in evaluating the examination.

Doubilet and Mulholland regarded spasm of the distal end of the common duct at the sphincter of Oddi as physiologic, its chief importance being increased intraductal pressure causing reflux of bile into the pancreatic duct. They proposed this as one of the chief factors in the production of pancreatitis, and recommend sphincterotomy for relief of such excess pressure, together with cholecystectomy whether or not the gallbladder is normal.

There is variance of opinion as to whether sphincteric disturbances are entirely physiologic. Cattell and his co-workers regarded partial sphincteric obstruction as due to inflammatory stenosis which is associated with the formation of common duct stones in some cases. The complexity of the clinical picture of common duct stenosis was emphasized by the difficulty in recognizing this condition either before or after operation. Cholangiographic evidence of a ductal diameter of from 8 to 15 mm. is regarded by them as indicative of partial block, while a diameter over 15 mm. is presumed to be definite evidence of sphincteric obstruction, as is prolonged opacification of the duct. An exception to this is made when the gallbladder retains the contrast material, thereby permitting prolonged ductal opacification by constant excretion. Their opinion is that fibrosis of the sphincter of Oddi leads to stenosis, and that this occurs without common duct stones and without previous operation. Distal choledochal and sphincteric inflammation also follows operative trauma of sounding. Stones impacted in the distal end of the common duct can produce similar changes.

A prominent cause of the post-cholecystectomy syndrome is inflammation of the distal end of the duct, particularly when associated with retained stones. Intravenous cholangiography provides helpful data by outlining the common duct in suitable patients. Walters remarked that efforts should be made to ascertain the condition of the common duct at operation. He regarded operative cholangiography as supplemental to thorough exploration.

Inflammatory choledochal fibrotic stenosis in association with duodenal ulcers was described by Partington. His 3 patients had marked symptoms of biliary tract disease in the presence of normal cholecystograms. In 2 typical attacks had been reproduced by morphine. Operative cholangiography confirmed the preoperative impression of partial obstruction at the distal end of the common duct. The gallbladder was normal in 2 and slightly thickened in 1 patient. Microscopic examination of tissue removed at transduodenal exploration of the sphincter of Oddi showed marked inflammatory changes, which accounted for the partial obstruction.

Inflammation in the extrahepatic biliary system involves other areas. Cystic duct stenosis sometimes can be suggested from intravenous cholangiograms by failure of visualization of the gallbladder in an otherwise normal study. Strictures elsewhere in the biliary system also cause obstruction. Here intravenous cholangiography is supplemented by either transhepatic or operative cholangiography, particularly when congenital strictures are suspected.

Lorinc and Herczeg mentioned that ducts measuring 20 to 30 mm. in diameter were not infrequent after cholecystectomies done 10 to 20 years before. They regarded this as a compensatory reservoir function of the common duct, even though others believe it to be a manifestation of biliary dyskinesia.

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PANCREATITIS

Pancreatitis is a relatively frequent disease which often is unrecognized. Its acute stages are dramatic, but the chronic form may be asymptomatic or produce relatively nondescript complaints. At present, obstruction at the distal end of the common duct is regarded an important causative factor because increased intraductal pressure favors reflux of bile into the pancreas. Other conditions also are associated with pancreatitis. Recently Dreiling and Janowitz summarized these and enumerated the experimental evidence for the production of pancreatitis. Basically, this is an infectious disease with an obstructive common channel mechanism the prime cause. Other factors include abdominal trauma, alcoholism and cirrhosis of the liver, metabolic and nutritional disturbances such as hemochromatosis, essential hyperlipemia, vascular diseases as seen with diabetic ketosis, periarteritis nodosa and certain toxic and allergic diseases. Cholecystitis and cholelithiasis occur together with pancreatitis in about 70 per cent of cases. Peptic ulcers perforating towards the pancreas often are associated with pancreatitis. Doubilet and his co-workers stressed the association between physiologic sphincteric spasm, increased intraductal pressure and biliary reflux. The hereditary aspects of chronic pancreatitis were discussed by Gross and Comfort, and Comfort and Steinberg.

Chronic pancreatitis often appears after previous acute episodes, but may occur without such a preexistent process. Parenchymal and intraductal calcareous deposits often are present with chronic pancreatitis. The intraductal stones, as a rule, are discrete, whereas parenchymal stones are either diffusely disseminated or localized, with adjacent chronic inflammatory changes. The clinical activity of chronic pancreatitis cannot be directly correlated with pancreatic lithiasis. Nevertheless, their demonstration radiologically often points towards a correct diagnosis.

Pancreatitis occurs in children as well as adults. Reeves and Moran described diffuse pancreatic calcification in a 4-year-old girl who had no pain. They noted 5 other cases in patients less than 35 years old, all with long illnesses. Blumenstock, Mithoefer and Santulli reviewed 36 cases from the literature. These were usually acutely ill patients with generalized abdominal pain, nausea, vomiting and tenderness. These authors presented 4 additional cases in infants, 3 being 15 months or younger, and the fourth 6 years old. A definite association with severe abdominal trauma was present. Parasitic infestations of the intestinal tract and abnormalities of the biliary system were relatively frequent.

The association of alcoholism and acute pancreatitis is important. Bockus and his co-workers found that the incidence of biliary disease and alcoholism were about equally important in this respect.

The clinical picture of acute pancreatitis is varied, and often is misdiagnosed as acute biliary tract disease, intestinal obstruction, myocardial infarction or acute appendicitis. Jaundice is relatively common in patients with underlying gallbladder disease, but is less so in alcoholic patients. Laboratory evidence of serum amylase elevation is helpful. This may be misleading, since similar changes are encountered in other conditions.

Acute pancreatitis occurs as suppurative pancreatitis with abscess formation, acute edematous interstitial pancreatitis, and necrotic pancreatitis with hemorrhage. These cannot be accurately distinguished one from another clinically or radiologically. Occa-

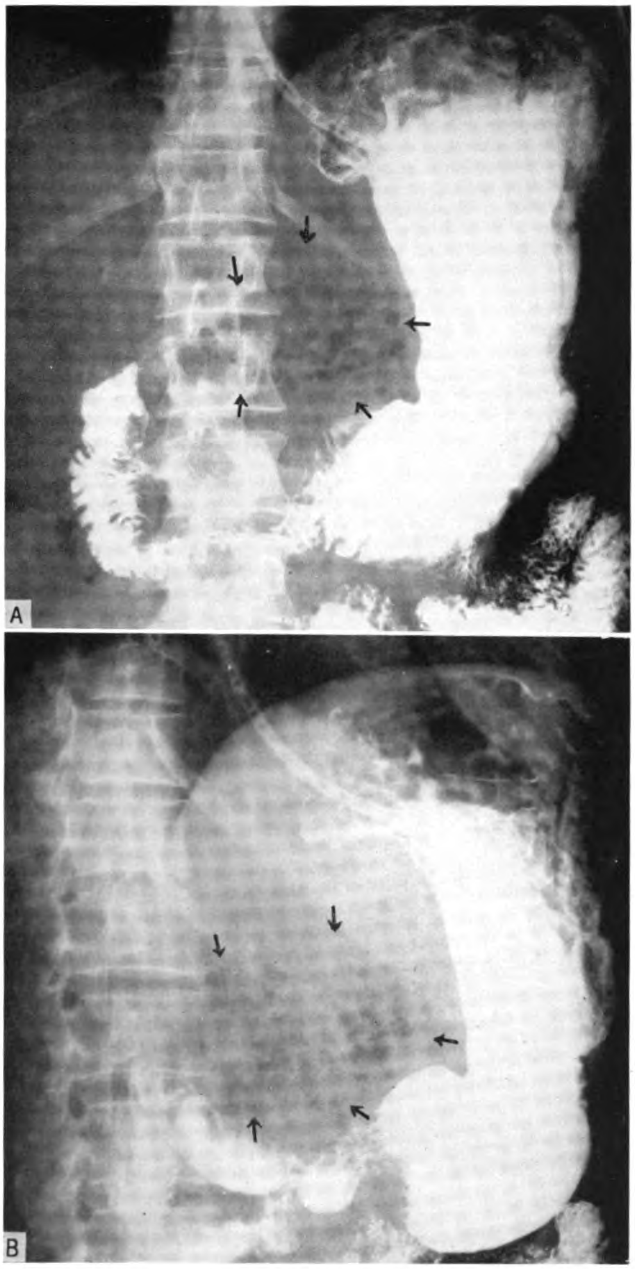

FIG. 178.—*A*, Acute necrotizing pancreatitis associated with penetrating duodenal ulcer. The gas shadows are in the head and body of the pancreas (arrows).

B, Same patient, oblique view. (Courtesy of Dr. Paul Steinhorn, Mid-Island Hospital, Bethpage, N. Y.)

sionally, a gas-containing pancreatic abscess is distinguished by locules of gas in the pancreatic bed which are not in continuity with the stomach or bowel. In other forms the changes produced are due to localized or diffuse swelling which produces pressure changes on contiguous organs, particularly the stomach and transverse colon. Acute pancreatitis also is associated with ileus which may simulate intestinal obstruction. Sometimes a sentinel loop of distended small bowel appears in the left upper abdomen.

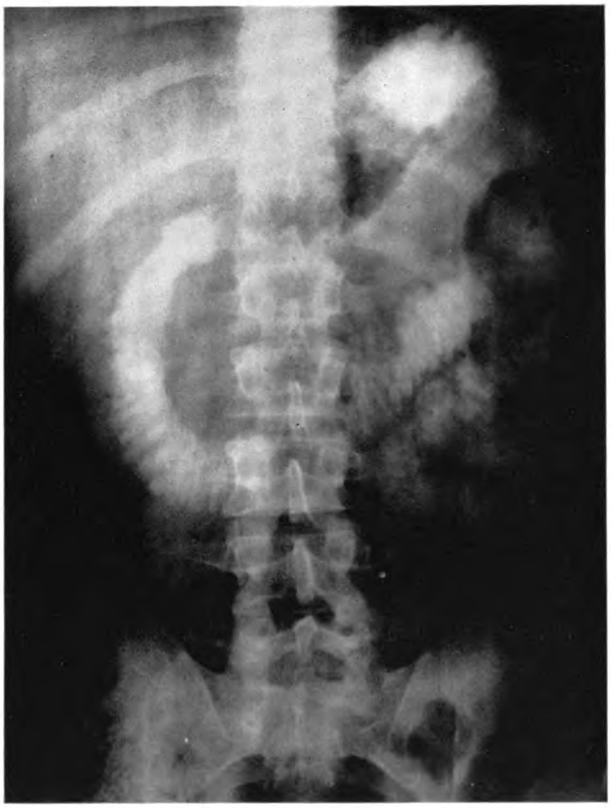

FIG. 179.—Acute pancreatitis in a 28-year-old man with acute epigastric pain radiating to the back and flanks. Jaundice, fever and elevated serum amylase were present. Film after ingestion of 60 c. cm. of 75 per cent Hypaque shows widening of duodenal loop and smoothening of its inner aspect.

This is not a specific sign, but together with a good clinical history, has some merit.

The radiologic changes produced by continuity involve the duodenal sweep either as a flattening of the inner aspect of the second portion, or with marked edema, the lesion simulates a cystic mass within the loop. Flattening of the greater curvature of the stomach was seen by Isaacs and Epstein in a case of acute pancreatitis verified at operation (Fig. 182). With recession of the acute phase, calcification appeared and the gastric deformity vanished. Stuart reported that with acute pancreatitis the segment of transverse colon near the head of the pancreas is devoid of gas, while both the hepatic and splenic flexures are well outlined. Paxton and Payne reviewed the plain films of 125 patients with acute pancreatitis. Eighty-two revealed segmental ileus involving the stomach and lesser and large bowel. These authors noted that the transverse colon showed this more often than the rest of the bowel, and considered such a change suggestive of acute pancreatitis.

In reviewing our own material, we found that the presence of sentinel loops was not particularly helpful. Ileus was quite pronounced. In some the colon was dilated, while in others the absence of gas from the large bowel suggested the possibility of small intestinal obstruction. Other signs included deformities of the duodenal bulb, haziness of the left psoas shadow, and left basilar pleural effusion.

The association of non-visualization of the gallbladder with acute pancreatitis is well known, and sometimes this is misleading. In 3 recent cases the gallbladder function returned within a week after the acute

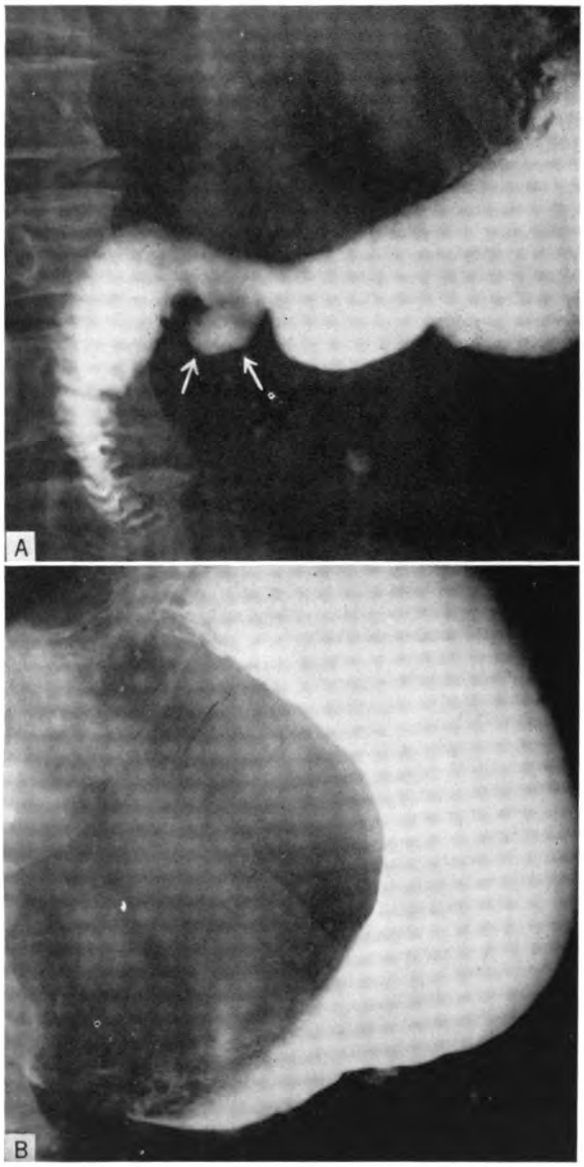

FIG. 180.—*A*, Acute hemorrhagic pancreatitis with excavation into head of pancreas in a 67-year-old man. Barium extends into this cavitation (arrows).

B, Same patient, 2 weeks later. The duodenal sweep has narrowed, and some barium is extrinsic to the distal greater curvature. A large collection is present posterior to the stomach. At operation a large abscess cavity was found between the stomach, liver and anterior abdominal wall, extending from a large indurated pancreas. Fat necrosis and free fluid were present.

episode subsided. In one the gallbladder was normal, but in the other two stones were present. Kaden, Howard and Doubleday remarked that a cholecystogram during the first week of illness with acute pancreatitis was of assistance only if the sac visualized. They observed return of function within a month after subsidence of acute pancreatitis. Silvani and McCorkle reported 28 cases of acute pancreatitis with intravenous cholangiography examinations. The gallbladder visualized normally in 12, and in 6 visualiza-

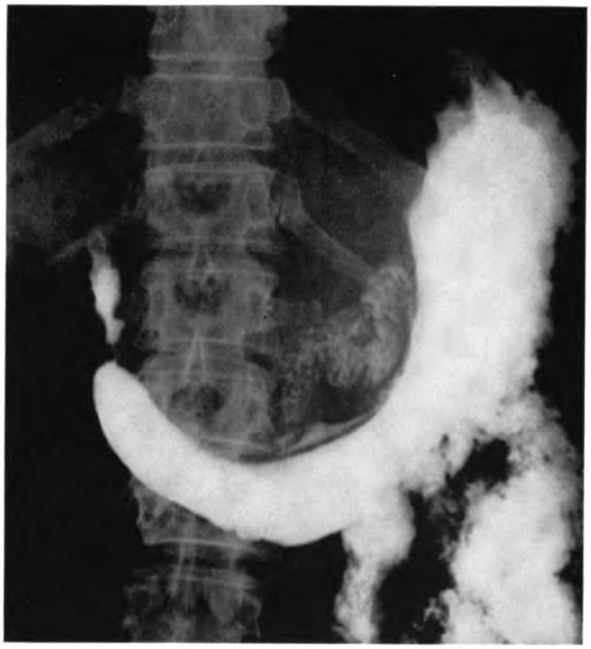

FIG. 181.—Indentation into the lesser curvature from an enlarged pancreas. At operation acute pancreatiti involving mainly the body of the pancreas was found.

tion returned after the pancreatitis subsided. Five patients operated upon had normal gallbladders. No evidence of liver function impairment was obtained in these patients, nor was jaundice encountered.

Many patients with chronic pancreatitis have complaints which are so variable that it is almost impossible to make a definite clinical diagnosis. The demonstration of pancreatic calcifications is useful in evaluating symptoms. A considerable degree of fibrosis of the pancreas may be associated with diabetes and steatorrhea, and pancreatic cysts are relatively common. Jaundice appears occasionally when a stone impacts into the pancreatic duct and occludes the common bile duct. Poppel noted an increase in the size of the papilla of Vater due to edema from chronic relapsing pancreatitis. In severe episodes the edema spreads to the contiguous mucosa a sign regarded as indicative of a relapse. A similar change has been observed in the presence of regional inflammation. In late cases with periduodenal scarring the loop becomes irregularly distorted, and sometimes is obstructed.

Non-visualization of the gallbladder was observed in 5 out of 20 patients with chronic recurrent pancreatitis by Maimon, Kirsner and Palmer. In 3 of the 4 patients with non-visualization operated upon, no biliary tract disease was found. Cholelithiasis was found at operation in 2 of 3 other patients who had normal cholecystograms.

Occasionally a solitary stone of doubtful origin appears in the duct of Wirsung. Johnson and Baker, in a report on this condition, noted that such solitary stones without clinical evidence of pancreatitis, may be treated by removal of the stone.

Visualization of the pancreatic ductal system has been accomplished by Doubilet and Poppel by operative intubation and injection of contrast material. Dilatation of the ducts, abscess and cyst formation were thus demonstrated in patients with chronic pancreatitis. Doubilet and Mulholland were able to intubate the pancreatic duct in a fairly large proportion of their cases. However, Walters encountered considerable difficulty in identifying this structure at operation, even after an incision up to a centimeter in length had been made through

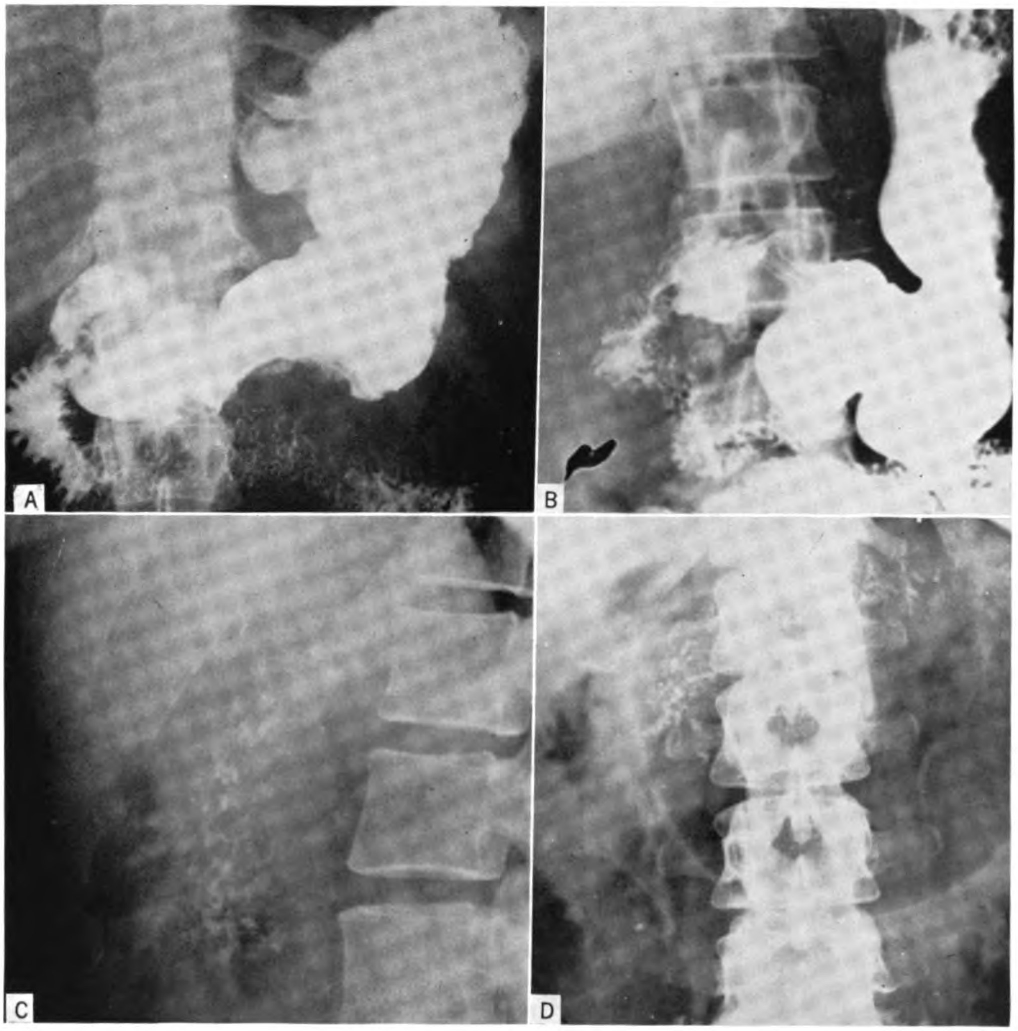

FIG. 182.—*A*, Impression into greater curvature of stomach due to acute pancreatitis, verified at operation. *B*, Same patient, 10 months later. The deformity has cleared.
C and *D*, Same patient, sixteen months later. Diffuse pancreatic calcificration has appeared. (Epstein B. S. and Isaacs, I., courtesy of Radiology, *51*, 214, 1948.)

the duodenal mucosa overlying the sphincter of Oddi. He commented on the great variability in the normal anatomy of the pancreatic ductal entrances into the common duct.

The effects of acute pancreatitis on adjacent structures often are extensive. Exudate containing pancreatic juice may enter the lesser sac through the foramen of Winslow, producing a chemical peritonitis and consequent accumulation of fluid. Inflammatory changes also may appear over the dome of the liver and down the right lumbar gutter, producing a picture simulating acute cholecystitis. Sometimes the stomach or duodenum becomes inflamed, with gastrointestinal bleeding. A retroperitoneal spread may be followed by abscess formation. Pancreatic juice extending down the sigmoid mesocolon or upwards into the subphrenic spaces may cause collections of fluid and abscesses. Similar changes have been noted rarely along the aorta or the mediastinum. The left perinephric space may be entered, simulating a perinephric abscess. Extension to the porta hepatis may result in fibrotic changes in the distal end of the common duct, or in the adjacent gallbladder or duodenum.

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OPERATIVE AND POSTOPERATIVE CHOLECYSTOGRAPHY AND CHOLANGIOGRAPHY

Investigation of the gallbladder and biliary ducts at operation is an important procedure

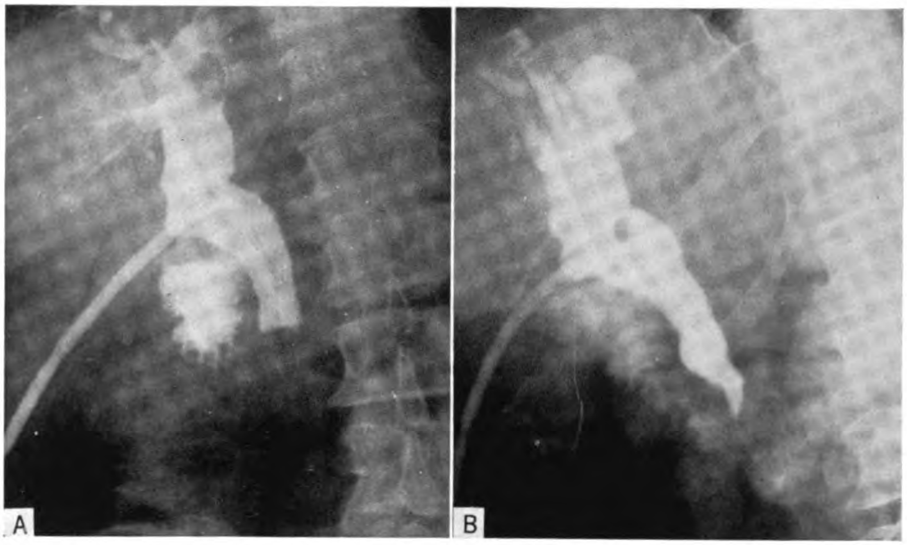

FIG. 183.—*A*, Spasm at distal end of common bile duct, "T" tube cholangiogram. Note the blunted end of the duct. Contrast material has entered the duodenum.

B, Same patient, about 5 minutes later. The spasm has relaxed and the distal end of the duct is visualized.

for the diagnosis of ductal patency or the identification of retained stones. Smith, Engel, Averbrook and Longmire noted that the incidence of retained stones in the common bile duct after negative explorations was about 4 per cent, with a range of from 3 to 10 per cent after all common duct explorations. Thomson commented that residual or overlooked stones were reported to remain in from 10 to 25 per cent of all common ducts explored.

Postoperative cholangiography is accomplished by the injection of contrast medium into an indwelling "T" tube. This may be done just before the abdomen is closed, or a few days later. As an operating room procedure, it affords a last chance for the identification of intraductal disturbances. Care must be taken to avoid the introduction of air bubbles. In evaluating a "T" tube cholangiogram, films should be made with some degree of overexposure. In some cases body section radiography is helpful in identifying retained stones.

The return of symptoms after cholecystectomy often makes reexamination of the biliary tract necessary. The evaluation of intravenous cholangiograms for this purpose sometimes is difficult. Not every patient with a post-cholecystectomy syndrome will have good visualization of the common duct either by intravenous or reinforced oral cholangiography. Our proportion of failures is about 15 per cent in cases anticipated to have a good visualization. Nevertheless,

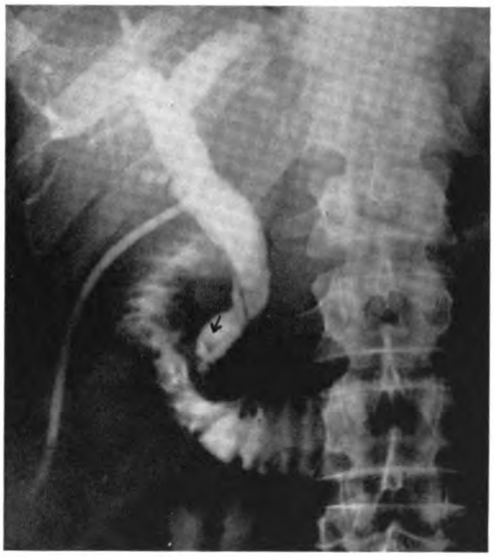

FIG. 184.—"T" tube cholangiogram reveals a triangular shaped radiolucent stone (arrow) in the distal end of the common duct with no obstruction.

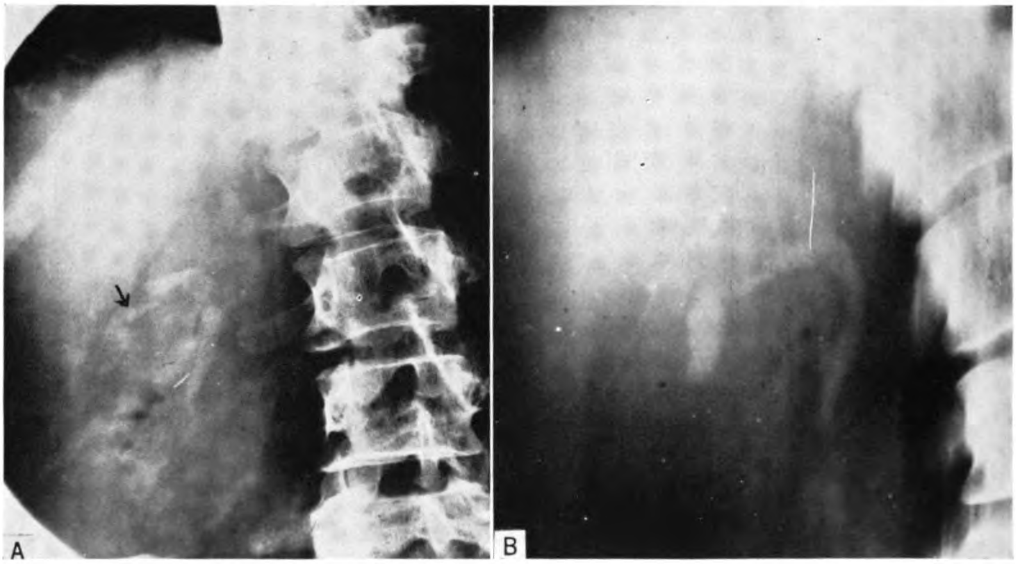

FIG. 185.—*A*, Intravenous cholangiogram. 40-minute film. The common duct is well opacified. A dilated, elongated cystic duct is visible (arrow), and proximal to it is a sac-like structure.

B, The body section film clearly shows the dilated sac-like structure. At operation a dilated cystic duct with cholecystitis was found.

opacification of the common duct is most helpful in the identification of radiolucent gallstones. Reformation of a gallbladder usually means dilatation of a large cystic duct stump. Occasionally this is a cause for recurrent symptoms, and stones may form in these recurrent sacs.

Filling of the pancreatic duct during retrograde cholangiography is relatively frequent, but similar occurrences during intravenous cholangiography are rare. Howell and Bergh found elevated serum amylase values in 25 out of 27 cases in which pancreatic reflux followed cholangiography, and observed that this paralleled the degree of pressure used for the injection. However, there is no evidence either in the literature or in our experience that such reflux is followed by pancreatitis.

The interpretation of postoperative cholangiograms requires consideration of the filling of the ductal system, its distribution and caliber, the presence of retained stones or other filling defects, and the capacity for emptying. Most of the stones seen on these examinations are radiolucent, but occasionally a calcified stone is found. Spasm of the distal end of the common duct may occur in patients who have no obstructive changes. Sometimes the passage of the contrast material into the duodenum is delayed momentarily, or, occasionally, a delay of 5 minutes or longer is noted before the material passes into the duodenum. If spasm is suspected, the use of nitroglycerine under the tongue, or the instillation of cyclaine into the "T" tube may be helpful. An evidence of spasm seen repeatedly by us is a transverse appearance of the distal end of the duct about a centimeter or two above its termination at the sphincter of Oddi. When the spasm relaxes, the distal common duct assumes a tapered appearance. Occasionally a semicircular indentation appears in the inner wall of the duodenum as a transitory phenomenon. This may represent a passing muscular reaction, and no clinical import is attributed to this change.

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EFFECTS OF SURGERY ON THE DIGESTIVE TRACT

The untoward effects of surgery on the digestive tract may be divided conveniently into two main categories, those due to procedural errors and those incident to anatomic, inflammatory, altered physiologic, and mechanical changes. These include fistulous tracts, intestinal pouches and rotational deformities of intestinal loops, exclusions of various loops, and the manifold effect of adhesions.

Mechanical effects of operative procedures on the stomach sometimes are seen at the site of anastomosis. In days past, when gastroenterostomy was the procedure of choice for duodenal ulcer patients, mechanical obstructions at or near the stoma produced pronounced dilatation of the afferent loop of jejunum and the duodenum. Often this was accompanied by vague abdominal complaints, and sometimes the dilated bowel was palpable in the mid-abdomen. Obstructions at the stoma were associated with pronounced gastric retention, with the consequent train of symptoms.

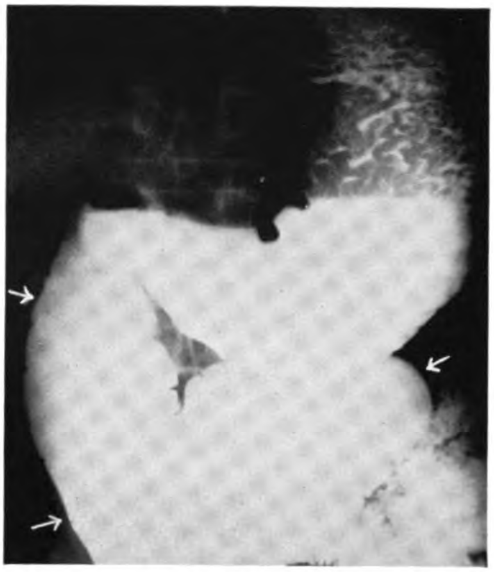
FIG. 186.—Marked dilatation of duodenum (arrows) 29 years after gastrojejunostomy for peptic ulcer.

Obstruction of the afferent loop after subtotal gastrectomy and gastrojejunostomy is accompanied by vague and intermittent distress, sometimes with regurgitation of bile. Afferent loop obstruction is ascribed to rotation of a relatively long proximal jejunal loop behind the distal loop, resulting in angulation and torsion of the jejunum at the point of attachment to the lesser curvature. This complication is relatively uncommon, Quinn and Gifford reporting its occurrence only 6 times in 500 resections for ulcer. Wells and MacPhee reported the afferent loop syndrome as occurring in 18 per cent of 239 cases following subtotal gastrectomy, 10 per cent being regarded as severe. These authors noted that the symptoms may disappear within 3 months after operation. If they persist longer than this time, a true afferent loop syndrome is considered present.

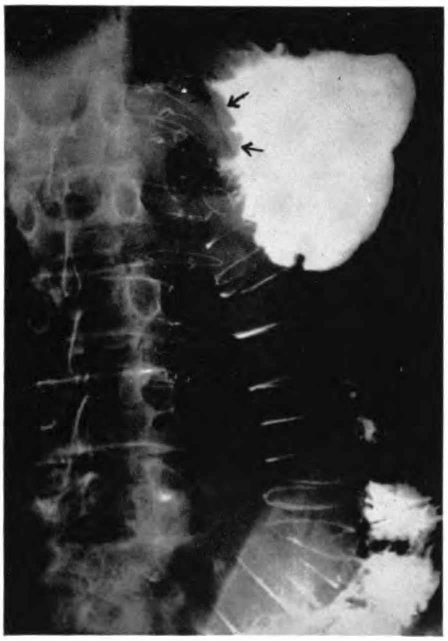

FIG. 187.—Obstruction of afferent loop in a 59-year-old man 6 years after subtotal gastrectomy for peptic ulcer. The distal end of the stomach is infiltrated with carcinoma (arrows), which blocked the stoma. The parallel rings represent dilated valvulæ conniventes.

Afferent loop obstruction may give rise to relatively severe pain simulating pancreatitis. Such a case occurring 4 months after subtotal gastrectomy was reported by Thal and Perry. The serum amylase levels in this patient were elevated, so that the true nature of the disease went unrecognized. At necropsy the epigastrium was occupied by a greatly distended obviously gangrenous and perforated duodeno-jejunal loop, with abscesses between the duodenum and the anterior abdominal wall. The pancreas was normal. The radiologic identification of afferent loop obstruction may be difficult unless barium enters the involved area. If it does not, and no opacity is visible to suggest its presence, the diagnosis must be made on clinical grounds alone.

Obstruction of the efferent loop after subtotal gastrectomy also gives rise to a confusing clinical picture characterized by symptoms referable to hypersecretion and hyperacidity, with epigastric fulness, nausea and vomiting. Obstruction of the efferent aspect of the stoma occurs soon after operation, either on a mechanical or functional basis. Mechanical factors include technical difficulties or the development of adhesions. If pure mechanical obstruction is present, the probability is that the disturbance affects both the afferent and efferent loops and the stoma itself. With functional disorders there may be gastric retention over a variable time. With subsidence of inflammation and edema, symptoms often disappear. This condition appears more often in elderly patients with low serum protein. Potassium depletion and sodium retention also may be factors.

Gastroileostomy may be performed in error. Severe weight loss, pain, diarrhea, the passage of undigested food in the stool, some fecal vomiting and bleeding may appear soon after operation. In a 50-year-old man seen by us, symptoms appeared about 10 weeks after subtotal gastrectomy for duodenal ulcer. Severe vomiting was the outstanding symptom, together with rapid weight loss. The diagnosis was suggested radiologically when the contrast material passed almost immediately into the distal ileum and colon. Gastric refilling may appear in patients with gastroileostomy, the barium passing through the pylorus to the ileum, and then reentering the stomach. Under such circumstances a film made 1 or 2 hours after a barium meal shows the stomach empty, while the 5-hour film again reveals barium in the stomach.

Gastrojejunocolic fistulas may be indicated by diarrhea, wasting and fecal vomiting. The physical condition of these patients varies greatly, some being in relatively good condition and others emaciated. This complication usually follows subtotal gastrectomy, but also is observed with colonic neoplasms which become affixed to small intestine. The preferred method for making this diagnosis is barium enema. It is possible for these tracts to be one-way passages, with the contrast material bypassing the fistulous tract when ingested by mouth.

Jejunogastric intussusception occurs as a relatively late complication of gastroenterostomy or gastrectomy, either or both jejunal loops entering the stomach through the stoma. This is accompanied by midepigastric pain, persistent vomiting and hematemesis, and sometimes a palpable mass. It may occur acutely, or less often in a chronic form with relatively little discomfort. Radiographic examination discloses this condition by the presence of small bowel within the stomach. The size of the defect depends upon the extent of invagination. Compression studies are useful in identifying the characteristic striations of the lesser bowel within the lumen of the stomach.

Postoperative volvulus of the small intestine presents the picture of intestinal obstruction. A single loop of bowel may be involved, or the entire small intestine can rotate. The later is more frequent in children, usually associated with a congenital mesenteric fold deformity. In adults volvulus occurs around a postoperative adhesion, underlying mesenteric attachment abnormalities being less frequent. A blind loop of bowel may rotate and present a bizarre clinical picture, inasmuch as the loop is not in continuity with the remainder of the intestinal tract. Such a case of volvulus of a blind ileal loop following ileosigmoidostomy was reported by Eliasoph, Marshak and Wolf. Colonic voloneus also may occur with signs and symptoms of an acute obstruction.

Intestinal obstruction also follows intra-abdominal adhesions, which may form soon after operation, or may become symptom-producing much later. This has been discussed before. Adhesions sufficient to cause a mechanical block can appear within a few days after operation. The distinguishing features between this condition and physiologic ileus are indeterminate but the diagnosis may be facilitated by the use of water-soluble contrast substances such as Hypake or Gastrografin. Internal hernias also may form because of adhesive bands, or may follow rents in the mesentery.

An interesting complication of gastrointestinal surgery is the development of blind pouches. After gastroenterostomy, local outpocketings result from the weight and pull of the jejunal loop on the stomach. Landy suggested that these might be an etiologic factor in the production of stomal ulcers because of the prolonged action of acid gastric juice on the stoma of the pouch. In 2 cases described by Thommen, these lesions had been called ulcers, but at operation were found to be simple deformities caused by the traction of adhesions. Intestinal pouches of the blind end also follows side-to-side anastomsosis of the small and large bowel. Heifetz and Senturia reported 8 cases, 5 with symptoms.

Marked dilatation of an excluded loop of intestine sometimes follows ileotransversostomy. In a case seen by us after operation for regional enteritis the ascending and proximal transverse colon became about 10 times normal size, and retained barium for more than 6 months. This patient complained of an unpleasant sensation of weight in the abdomen. Sentoria and Heifetz noted symptoms simulating partial intestinal obstruction and macrocytic anemia among the complications of intestinal pouches following lateral anastomosis.

Physiological disturbances after subtotal gastrectomy, referred to as the "dumping syndrome," are characterized by epigastric fulness, distress, pallor, nausea, palpitation and vomiting among other systemic complaints, and occur at varying times after operation. Goligher and Riley noted no definite relation to the type of operation, but observed that it was slightly more frequent after extensive resections. In many, spontaneous amelioration of symptoms was observed. Webber, Bender and Moore recently reviewed the various etiologic concepts and concluded that the physiological alterations were due to the altered anatomy. Distention of the small intestine is considered an important factor. The pyloric sphincter is lost after gastrectomy, and food passes immediately into the upper jejunum or duodenum by gravity. This distention is held responsible for untoward symptoms. Symptoms may be reproduced by administering hypertonic glucose as a test substance. Patients given 150 cc. have similar symptoms at a time corresponding with that associated with eating. The introduction of hypertonic solutions produces an influx of body fluids into the jejunum, distending this organ with consequent symptoms. Similar results were obtained after introduction of balloons into the jejunum and inflating them.

However, not every patient so examined presents clear correlations, and probably there are many other factors still to be understood. The radiologic changes associated with the dumping syndrome are not diagnostic.

The influence of vagotomy on gastrointestinal activity is manifested chiefly by reduction in peristaltic activity and atony, insofar as radiologic changes are concerned. There is a tendency for gradual remission of this effect over a prolonged period of time.

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INFLAMMATORY DISEASES OF THE SPLEEN

Splenic abscesses usually follow splenic artery or vein thrombosis, or infected emboli produce infarcts which subsequently break down. They were seen more often in preantibiotic days, and even then were infrequent. Occasionally perisplenitis results in fixation of the spleen against an adjacent viscus such as the stomach, and causes secondary pressure defects.

A case of primary splenic abscess was reported by Gelfand. The dome of the diaphragm was elevated, and a fluid level was seen beneath the left diaphragm displacing the heart upwards. Fourteen pints of water brown pus were aspirated.

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PARASITIC INFESTATIONS

Amebiasis.—The most frequent protozoan infection affecting man is Entamoeba histolytica. This primarily affects the colon, and secondarily involves the liver and lungs. Extension into the thorax isfrom the underlying liver or diaphragm, but occasionally pulmonary involvement occurs as an isolated phenomenon.

The parasite passes through two phases, the trophozoite and the cystic stages. The active trophozoites emerge from cysts which pass to the ceum after being swallowed. These are ingested with contaminated food or water, and may also be transmitted by flies or infected food handlers. Diarrhea with blood and mucus is common in the acute phase, acute amebic dysentery. The chronic gastrointestinal disturbances of variable diarrhea, nausea, vomiting and abdominal pain are more common.

Amebic colon ulcers tend to concentrate in the cecum and ascending colon, but may involve the entire bowel and extend to the terminal ileum. These vary considerably in size. The larger ulcers destroy the submucosa and extend laterally as flask-shaped craters, with intact mucosa between them.

Liver abscess is the most frequent complication. This can occur insidiously at any time, with no indication of the underlying amebiasis. Fever, pain in the liver area and sometimes in the chest are the presenting symptoms. While liver abscesses usually are unilocular in the right lobe, multiple abscesses are not infrequent. An inflammatory reaction develops with chronic, and at times, acute hepatitis. With extension of the inflammation to the diaphragm, an amebic abscess may pass into the chest.

In the asymptomatic stage, the patient may become a cyst carrier, with confusing minor gastrointestinal disturbances. Episodes of diarrhea and constipation are not uncommon. The symptomatic stage of the disease is reached when the cysts break through and the bowel inflammation becomes marked.

The radiologic diagnosis of early amebiasis is difficult. The usual picture is one of hypermotility of barium through the small intestine and rapid passage into the colon. No gross mucosal alterations are visible, but there may be considerable cecal irritability, which in itself is not diagnostic of anything specific. During the acute phase, cecal spasm and irritability becomes more pronounced, and as the inflammation increases the cecum and ascending colon are rendered into a cone-like structure. The cecum becomes granular and irregular. With further progression to a chronic stage, the ascending and sometimes the transverse colon develop localized narrowings and irregularities associated with spasm. In the most extensive form, the entire colon is markedly irritable.

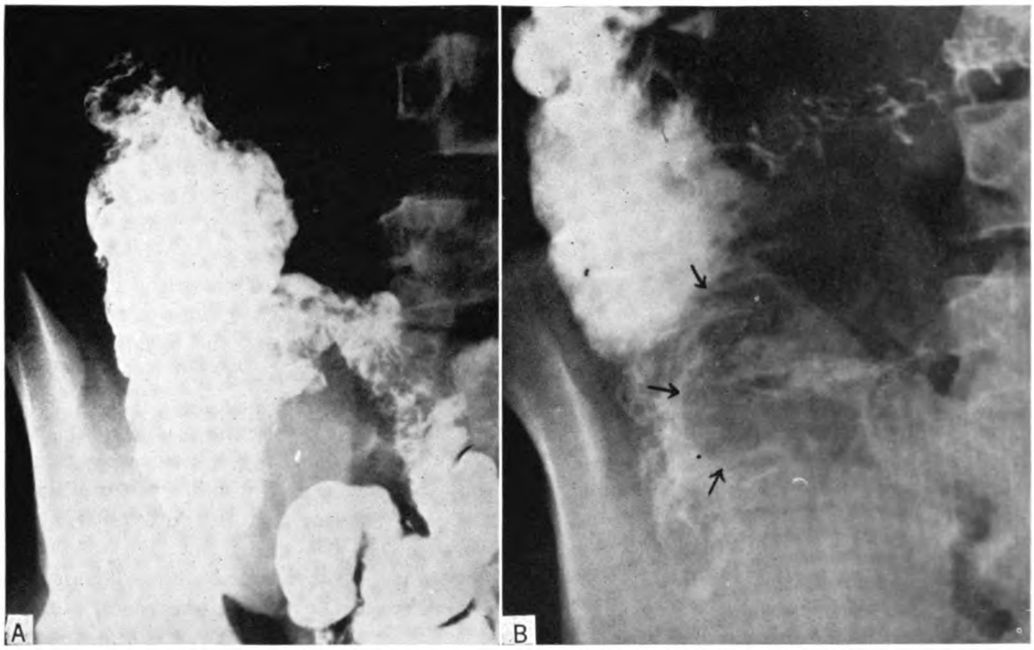

FIG. 188.—*A*, Amebic cecitis in an 18-year-old male with pain and tenderness in the right lower quadrant. Amebæ found in stool. The cecum has a slight indentation in its medial aspect. The terminal ileum is not altered.

B, Post-evacuation film shows marked irritability of the cecum and edema of the ileocecal valve (arrows).

Localized granulomatous lesions of the cecum, and occasionally of the ascending and transverse colon, form amebomas. These not infrequently are associated with a more acute history and bloody diarrhea. A mass simulating a malignant growth is frequently present in this stage. In the late stage, the colon becomes tubular, much the same as with nonspecific ulcerative colitis.

Golden and Ducharme noted that amebic lesions usually were multiple, whereas neoplasms were single. Obstruction by amebomas tends to be partial, with the involved loop longer than that usually seen with neoplasm. Transition from the pathologic to normal mucosa occurs gradually with amebomas, and no spur formations appear. With carcinomas these changes are more apt to be abrupt. The response of amebomas to medical treatment is of distinct diagnostic assistance.

Hepatic abscesses reflect the underlying pathologic changes. With the appearance of a large purulent collection the right diaphragm becomes elevated and its motion restricted. A moderate pleural effusion frequently appears. The liver shadow itself may become enlarged.

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Toxoplasmosis.—This protozoan infection may develop as an intrauterine disease or as an early postnatal infection. The organism produces a vascular inflammation with necrosis of perivascular tissues, followed by granuloma formation and ending with calcification. The most frequent site is the central nervous system, with a classical picture of encephalitis with hydrocephalus. No direct effects upon the abdominal organs have been described.

Malaria.—The radiologic changes observed in malarial patients are those referable to splenomegaly, and occasionally hepatomegaly. No characteristic gastrointestinal changes are produced.

Metazoan Infestations.—Infection with flat worms produces both gastrointestinal and systemic disturbances. Of particular interest radiologically is infestation with taenia soleum, the pork tapeworm, taenia saginata, the beef tapeworm, and taenia echinococcus. These organisms have no gastrointestinal tracts, and consequently their radiologic identification is dependent on displacement of barium within the patient's intestine. Taenia echinococcus is too small to be so identified. However, in the early stage of infestation there is marked irritability of the intestinal tract. The pork and beef tapeworms reach considerable length, producing intraluminal ribbon-like defects up to 7 or 8 meters long. The adult worm is anchored in the jejunum and grows downward. The fish tapeworm, diphyllobothrium latum, has been identified radiologically, but these defects are smaller than either the beef or pork tapeworms.

In the cystic stage the pork tapeworm causes cysticercosis, identified by the presence of multiple, somewhat ovoid small widely scattered intramuscular calcifications.

Echinococcus infestation is widespread. Man acts as the intermediate host. After the tapeworm eggs are swallowed, the hatched embryos penetrate the intestinal mucosa, reaching the liver through the portal vein. These develop into hydatid cysts. If the arterial or lymphatic systems are invaded, echinococcus appears in almost any organ in the body. As a consequence of irritation, a fibrotic capsule is formed by the host around the capsule of the cyst. No anatomic communication exists between the enveloping capsule formed by the host and the hydatid cyst. Calcifications often appear in the wall of the host cyst. Perforation of infected liver hydatid cysts into the biliary tree permits entrance of the organism into the gastrointestinal tract. Perforation of liver hydatid cysts results in the spread of daughter cysts into the abdominal cavity, and a pronounced peritoneal reaction characterized by profuse serous effusion often occurs. These peritoneal cysts likewise become calcified. Primary abdominal cysts occurring in the mesentery or the omentum may be freely movable and quite large. Hydatid cysts also appear in the spleen, resulting in splenomegaly. These, too, occasionally are surrounded by calcification.

Schistosomiasis.—Infestation with schistosoma japonicum or mansoni gives rise to a marked inflammation of the liver as the larvæ enter the portal vein, with generalized abdominal discomfort, marked debilitation and diarrhea. There may be marked irritation of the gastrointestinal tract, especially the colon, as the eggs penetrate the intestinal wall. Following the acute stage a chronic stage appears, with large numbers of eggs carried to the liver, where they cause miliary abscesses which later appear as pseudotubercles. The diagnosis of this infestation depends upon the identification of the organism. It was recently pointed out by Rodriguez, Garcia, Palmieri, Rivera and Rodriguez-Molena that schistosomiasis of the

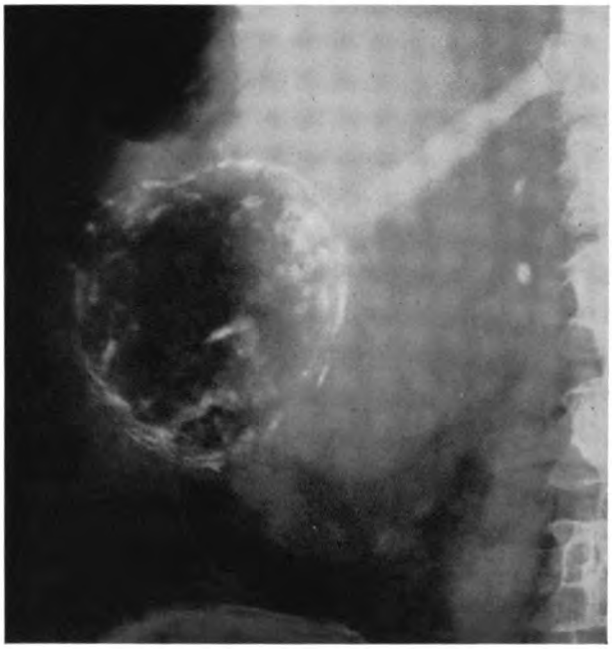

FIG. 189.—Echinococcus cyst in the liver of a 70-year-old man who had resided in Palestine for 30 years. Proven at operation 7 years before.

liver causes portal hypertension with marked splenomegaly and esophageal varices. Such portal hypertension may be relieved by shunting operations. If this is considered, splenoportography is of considerable importance.

The vesical type of schistosomiasis is due to infestation with the haematobium variety, and occasionally the mansoni. The radiologic diagnosis is sometimes made by the identification of diffuse somewhat parallel lines of calcification along the wall of the urinary bladder.

Nematode Infestations.—Infestation with the round worm, ascaris lumbricoides, is of interest to the radiologist. These are elongated, cylindroid worms usually present in the small intestine, where they may reach a length of over 30 cm. Inasmuch as they possess intestinal tracts, they may be identified by barium ingested by the worms. More often they are detected by virtue of their displacement of barium within the host's intestine, where the intertwined round worms produce a coiled appearance. These organisms have no perceptible effect on intestinal mobility. When sufficient bulk of worms accumulates partial intestinal obstruction results. This may occur in any age group. Sometimes the organisms are so prominent

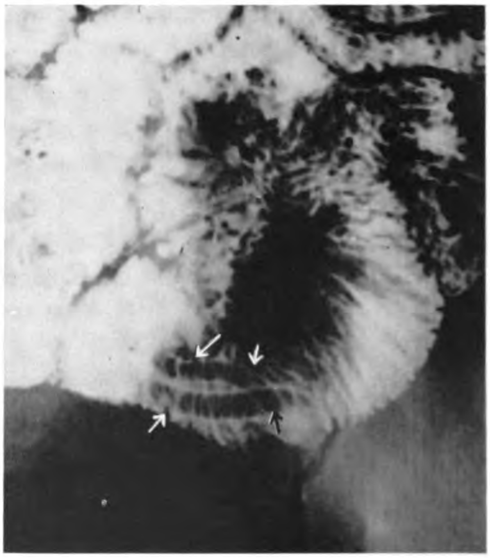

FIG. 190.—Ascaris in the lesser bowel (arrows).

that they are identified on scout abdominal films even when no contrast material is used. If obstruction occurs, the usual dangers of perforation, volvulus and strangulation all appear if the condition is not relieved. The association of acute pancreatitis with ascaris infestation in children was noted by Dobbs, who reported a case of a 12-year-old girl. In gathering 14 other cases of acute pancreatitis, 3 were found to have ascaris infestations. The pancreatitis is attributed to mechanical obstruction of the pancreatic ducts by the round worms.

Oxyuriasis.—Pinworm infestations are quite frequent, but rarely involve the abdomen. However, pinworms occasionally penetrate into the pelvic peritoneal cavity, producing a granuloma. Beddoe reported a 16-month-old girl with such a granuloma, recording this as the twentieth such case. At operation, the abscess contained egg and adult forms. The genital tract was considered the entering route. Extrinsic pressure of a peritoneal granuloma against the bowel causing pencil-like stools was reported by Symmers.

Hookworm Disease.—After penetrating the skin, the hookworm larvæ are carried to the lungs and enter the upper gastrointestinal tract after being expectorated and swallowed. Attachment of the hookworm to the mucosa is by means of hooklets, and this is followed by a local inflammation. Occasional instances of perforation of the intestinal tract and peritonitis have been recorded.

The earliest radiologic abnormality is a disorganized function of the lesser bowel, with excessive peristalsis, segmental contraction and mucosal distortion. Some jejunal narrowing occurs. These changes are attributed to injury of the intramural nervous system by the hooklet attachment.

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FUNGUS INFECTIONS

Fungus infections of the gastrointestinal tract are uncommon, actinomycosis being the most frequent offender. These organisms reach the enteric canal after being swallowed by patients who have bucco-pharyngeal lesions, a frequent manifestation of this disease. The stomach, duodenum and jejunum are not affected as a rule, but gastric involvement has been reported. Fuller and Wood described a 48-year-old man who had had postprandial heartburn, eructations and vomiting for 5 years. A deformity of the lesser curvature suggestive of an infiltrative ulcerating lesion was identified as being due to actinomycotic infiltration. Another patient with gastric actinomycosis was reported by Schmitt. A prepyloric rigidity suggesting a carcinoma was present, and at operation actinomycotic granulation with abscess formation and fistulas were found involving the anterior wall of the stomach, the transverse colon, the mesocolon and part of the omentum.

Actinomycotic infection of the cecum and appendix is more frequent. It is believed that the fluid contents of the small intestine do not break the continuity of the mucosa, so that the fungus cannot invade deeper tissues. With an attack of appendicitis, ideal conditions are provided for infection. The lesion then involves the cecum, the terminal ileum and the ascending colon with an inflammatory granulomatous mass, secondary infection is followed by sinus tracts, abscesses and fistulas. Further complications from adhesions produce obstruction. Radiologically, the lesion often is indistinguishable from tuberculosis, and may simulate enterocolitis or malignant disease. The diagnosis is established by demonstrating the characteristic sulfur granules from the lesion.

Colonic involvement with actinomycosis is rare. Hoffman reported a case diagnosed as diverticulitis with abscess formation and possibly neoplasm. This was regarded as the seventh in the literature, and in none had a correct diagnosis been made preoperatively. Fistula formation and secondary rectal stenosis is quite prominent with anorectal actinomycosis, which is considerably more frequent.

Intra-abdominal coccidiomycosis is rare. A case with peritoneal and small bowel involvement was reported by Greaves. This patient had no gastrointestinal symptoms. At necropsy, fungoid nodules were found studding the heart, spleen, kidneys, liver and peritoneum. Two intestinal ulcers were present.

Stiles and Davis mentioned that coccidiomycosis may give rise to ulcerating and nodular granulomatous lesions, and peritoneal dissemination may resemble tuberculosis.

Histoplasmosis of the abdomen and gastrointestinal tract is rare. A verified case of diffuse visceral histoplasmosis with antemortem findings of mucosal damage and early ulceration was reported by Beibert. This patient had a history of anorexia, weight loss, chills, loose stool, pain in the right lower chest, abdominal fulness and hepatosplenomegaly. Association of histoplasmosis with appendicitis and mesenteric adenitis was mentioned by Raftery, Trafis and McClure, who noted its presence in 5 per cent of cases of appendicitis and in 43 per cent of cases of mesenteric adenitis. Lymphoid hyperplasia was the predominant histopathologic finding. Inasmuch as histoplasmosis has an affinity for the reticuloendothelial system, the spleen is affected frequently. The diagnosis is suggested by multiple round calcific splenic densities. Some are solid, others have concentric or laminated appearances. These usually represent the healed stage of the disease, and cannot be distinguished from healed tubercles.

Abdominal infection with aspergillosis is unusual. This organism affects the lungs, and its presence in the gastrointestinal tract does not give rise to clinical complaints. We have encountered one case in which aspergillosis peritonitis was seen at postmortem examination in a 58-year-old patient who had been on intensive antibiotic and steroid treatment for polyarteritis nodosa. This patient had had previous gastrointestinal bleeding, attributed to cirrhosis of the liver. During treatment he developed bean-sized lumps on his scalp, under the chin and in the groin. One opened spontaneously, exuding a creamy yellow material from which no organisms could be cultured. Blood cultures also were normal. Radiologic examination of the abdomen disclosed a pronounced ileus, which was part of the picture of a severe peritonitis due to perforation of the lesser bowel. A pure culture of aspergillosis was obtained from the abdominal cavity.

Intra-abdominal involvement with blastomycosis is rare. Thompson, Sullivan and Fox reported a case localized to the cecum.

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LYMPHOGRANULOMA VENEREUM

Lymphogranuloma venereum is a viral disease of adult life, usually transmitted as a venereal infection. This is a systemic disease with acute and chronic inflammation of the regional lymph nodes. In males the infection is carried to the inguinal nodes, and in females the pelvic nodes are involved initially. Symptoms such as fever, joint pains and headaches point to the constitutional nature of the disease. The virus has been recovered from distant sites such as the cerebrospinal fluid as well as from the local lesions. The disease is of long duration, and has a poor prognosis. A positive Frei test is helpful in diagnosis.

Following invasion of the regional lymph nodes, retrograde lymphangitis and lymphatic stasis bring the infection to the rectal wall. At this time the patient complains of tenesmus, constipation and mucous and bloody stools caused by erosion of the rectal mucosa. The action of the virus on the rectal tissues, together with the lymphatic stasis, produces chronic ulcerating proctitis and periproctitis. From the rectum the infection extends upwards to involve the bowel in a continuous tubular or disseminated pattern. In some the lesser bowel is affected. Following rectal mucosal ulceration there is a fibrotic reaction, with pronounced perirectal thickening. Abscesses and fistulas then appear, and the bowel lumen is narrowed and tortuous. Fibrosis may become so extensive that a frozen pelvis develops.

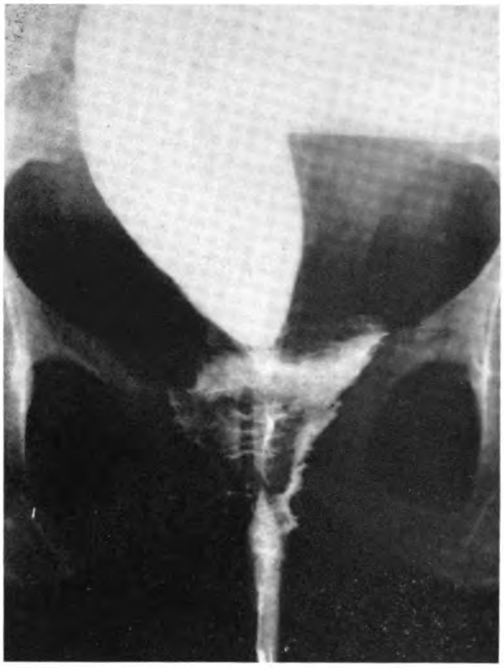

FIG. 191.—Vagino-rectal fistula in a 35-year-old woman with lymphogranuloma venereum. A rectal stricture was present 5 cm. above the anocutaneous junction. Note the vaginal mucosal folds.

The radiologic and pathologic changes of lymphogranuloma venereum are parallel, with marked rectal narrowing and stricture formation extending as high as 20 cm. into the bowel. Rectal strictures usually are located from 4 to 8 cm. above the mucocutaneous line. Perirectal and perisigmoidal sinuses and fistulas are frequent in the later stages, but rectovaginal or rectocolic fistulas are rare. We observed a 35-year-old woman with a rectal stricture 5 cm. above the anocutaneous junction who had a vaginorectal fistula diagnosed by barium enema (Fig. 191). Carcinomatous changes within affected areas have been reported.

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TUBERCULOSIS

Tuberculosis of the stomach and duodenum is infrequent, and usually is associated with ingestion of infected sputum. The stomach wall may be invaded by way of the blood stream, the lymphatics or from contiguous structures, particularly adjacent lymph nodes, the colon or the liver. In a review of the literature since 1917 Gaines, Steinbach and Lowenhaupt collected 49 cases including 3 of their own. Clagett and Waltman gathered 367 cases in the literature up to 1938. Gastric tuberculosis is usually associated with involvement of the adjacent lymph nodes, and this may dominate the picture. Sometimes the nodes are large enough to cause rather large filling defects in the stomach, or obstructive changes in the pylorus or duodenum. Gastrointestinal tuberculosis is the most frequent complication of untreated pulmonary tuberculosis, and the symptoms may mimic each other so that they cannot be told apart. However, diarrhea immediately points to the possibility of digestive tract involvement.

Tuberculosis of the stomach occurs as a combination of tumorous and ulcerative lesions, often indistinguishable from cancer. In some cases the crater resembles a benign gastric ulcer. Occasionally a preexistent gastric ulcer becomes infected with tubercle bacilli, as described by Gaines and his coworkers. Other forms include multiple small disseminated mucosal erosions and a miliary spread along the stomach walls.

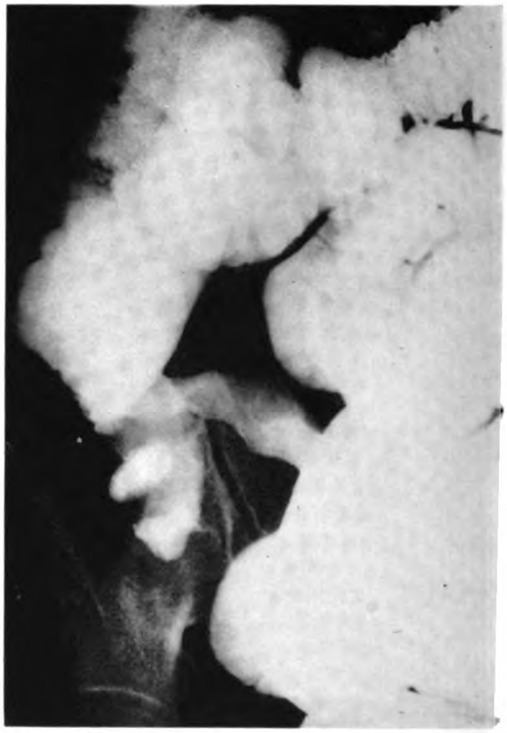

Fig. 192.—Ileocecal tuberculosis in a 28-year-olp woman with miliary tuberculosis. Mass felt in right lower quadrant.

The adjacent duodenum may be involved by pyloric tuberculosis. Rarely the duodenum alone is affected. A propensity towards fistula formation is associated with tuberculosis of the second or third part of the duodenum.

Infection of the jejunum and proximal ileum usually occurs as an ulcerative lesion. Lymphatic obstruction secondary to tuberculous adenitis may cause faulty absorption of intestinal contents, producing a radiologic picture of malabsorption with delayed motility, loss of mucosal markings and segmental dilatation.

The most common location of gastro-

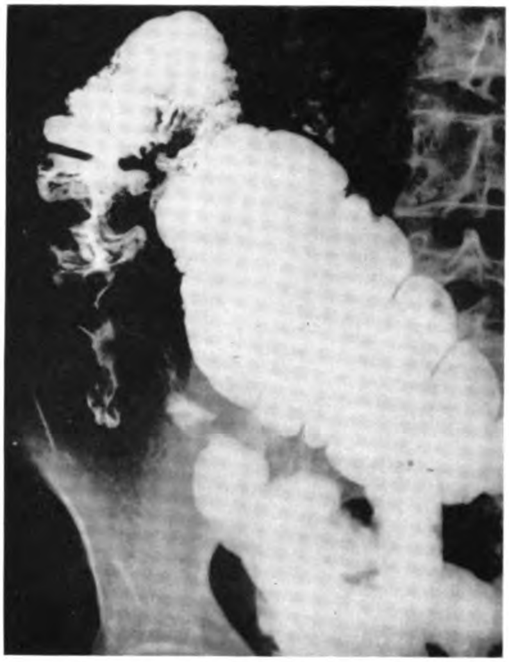

FIG. 193.—Ileocecal tuberculosis in a 47-year-old woman with abdominal cramps, diarrhea, weight loss and night sweats. There is marked irritability of the cecum, and the terminal ileum is narrowed and irregular.

intestinal tuberculosis is the ileocecal region. This occurs without specific complaints at times, but often is associated with symptoms like those of acute appendicitis or cancer of the proximal colon. Sometimes the lesion cannot be differentiated from a retroperitoneal tumor or regional enteritis. While ileocecal tuberculosis usually is associated with pulmonary infections, it may occur in its absence. The earliest radiologic changes are cecal irritability and relatively minor mucosal irregularity. Later granulomatous changes appear and the ileocecal valve becomes prominent, protruding into the cecum. Obstructive changes follow further inflammation and spasticity of the valve. Later scarring and incompetence permits free regurgitation between the colon and lesser bowel. The cecum becomes deformed, with grossly irregular mucosal folds. Multiple foci of inflammation are not uncommon, and in advanced cases the ascending colon and small intestine may be extensively involved. Occasionally skip areas of tuberculous colitis appear. The right psoas shadow may become hazy or disappear if pronounced lymphadenitis is present. The reversibility of these changes after adequate anti-tuberculous therapy was mentioned by Kogan and Janowitz, and by Brackin, Miller and Bell.

Tuberculosis limited to the colon is unusual. Such lesions usually are hyperplastic and may reach enormous proportions. In middle-aged people such granulomas are difficult to distinguish from carcinoma. As a rule, they are seen in young adults associated with tuberculosis elsewhere, but sometimes the primary lesion is hard to find. Tuberculous proctitis occurs in an ulcerated form, with numerous submucosal confluent tubercles. Occasionally, tuberculosis is limited to the rectum, producing an annular infiltration and a nodular mucosa.

Tuberculous peritonitis occurs as an acute or chronic disease. In the acute form the peritoneal surfaces are seeded by miliary tubercles as part of a generalized disease, or a relatively localized lymphadenitis may extend to the adjacent peritoneum by way of breakdown of a lymph node or rupture of a tubercle. This form also may proceed to a generalized process, with diffuse adhesions involving the entire peritoneal cavity.

Tuberculous peritonitis often causes an extensive ascites which is part of a generalized, rapidly progressive polyserositis. Sometimes only a scanty effusion is produced. The pathogenesis of effusion is obscure, but it is believed related to extensive lymphatic involvement. The response of tuberculous peritonitis to treatment has been commented on by Wichelhausen and Brown, and by Acheson.

The radiologic changes depend upon the extent of the peritoneal reaction, the creation of diffuse or local collections of fluid, and the effects of adhesion and tuberculous lymphadenitis. When a large quantity of fluid is present a diffuse haze appears over the abdomen, with varying degrees of ileus. With the formation of adhesions a relative fixation of the lesser bowel and colon is frequently associated with partial intestinal obstruction. No specific diagnostic sign for tuberculous polyserositis is recognized.

During the miliary stage of tuberculosis

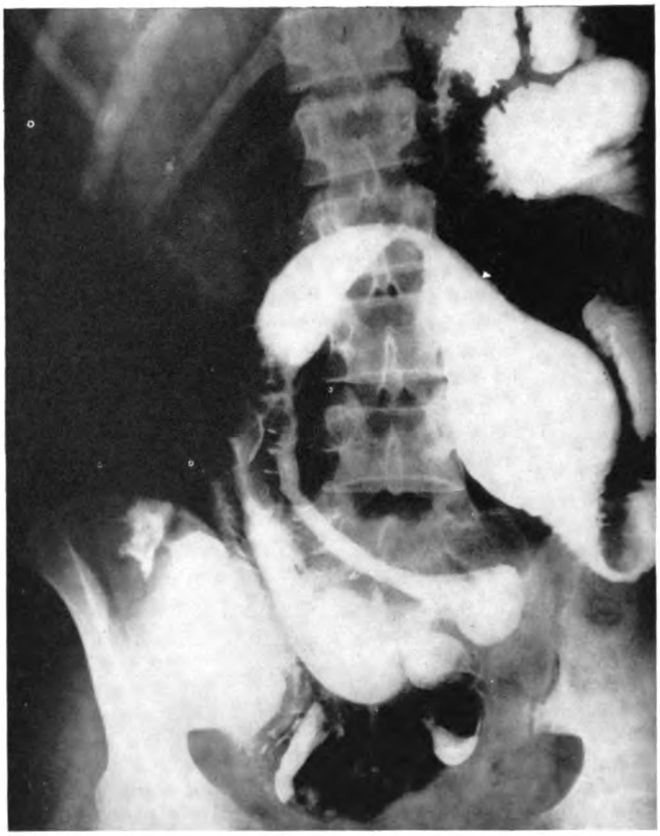

FIG. 194.—Tuberculosis of small intestine in a 25-year-old woman with vomiting, cramps, diarrhea and abdominal pain. Operation disclosed marked infiltration producing canalization of many segments of lower jejunal and ileal loops. Note the resemblance to regional enteritis and deficiency pattern.

the spleen may be seeded, and with healing these become calcified. Radiologically the calcified tubercles are visible as fairly uniform densities. These cannot be differentiated from splenic calcifications due to histoplasmosis.

Primary miliary tuberculosis of the liver was recently discussed by Terry and Gunnar, who reported 12 cases diagnosed by liver biopsy. These patients all recovered with adequate treatment. The *x*-ray examinations of the chest were negative in all. The patients were seriously ill, with abdominal pain, weight loss, toxicity, fever, lassitude, chills and sweats. No radiologic signs were associated with this condition.

Occasionally fistulas form between the intestinal tract and the bladder, uterus or the fallopian tubes. As a rule these cause no painful symptoms. Four cases of tubo-intestinal and one of tubovesical fistulas demonstrated by hysterosalpingography were described by Rozin. In these the lumen of the involved tube was enlarged and distorted. Later films showed the contrast material in the intestine. If the fistulous tract leads to the bladder, the lipiodol forms droplets which disappear after voiding.

Colorenal tuberculous fistulas occasionally are found, although these usually are due to perinephric inflammation or eroding renal calculi. Ellik and Getz pointed out that this diagnosis rests upon the demonstration of the tracts on retrograde pyelograms, the intravenous urograms and barium enema examinations being unreliable.

Rarely, caseating tubercles adjacent to or in the aorta cause aneurysms, which may go on to exsanguinating hemorrhages. Rothfeld, Garey and Twining reported a tuberculous fistula between the aorta and duodenum in a 58-year-old man who died from a profuse gastrointestinal hemorrhage. We saw a 62-year-old man who expired because of perforation of an atheromatous aortic placque containing a caseating focus into the third portion of the duodenum.

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SYPHILIS

Gastric syphilis is either a congenital or acquired disease. Both are uncommon. Mendl, Jenkins and Hughes found no more than 10 cases of congenital gastric syphilis in the world literature. One of their cases, a 10-year-old girl, had localized gastric syphilis of the middle of the greater curvature which progressed to typical linitis plastica, with deformity, rigidity, mucosal atrophy, absence of peristalsis and greatly diminished gastric capacity. A positive blood Wassermann reaction was obtained, and gastric biopsy confirmed the diagnosis. The condition responded to antisyphilitic treatment.

Acquired gastric syphilis is more common. The relative absence of symptoms in the presence of a markedly deformed stomach is noteworthy. The term gastric syphilis should be used only with tertiary lesions, inasmuch as the superficial gastritis sometimes seen in the primary and secondary stages is not specific.

Gastric syphilis originates as a small submucosal infiltration along the midportion of the greater curvature. The lesion increases in size, producing a mass with a crater and muscular infiltration. An hour-glass configuration is produced when the infiltration extends to the pars media and lesser curvature. As the lesion progresses to the antrum, a funnel-like stiffening simulating neoplasm appears. Involvement of the entire stomach results in the characteristic leather-bottle stomach. Meyer and Singer

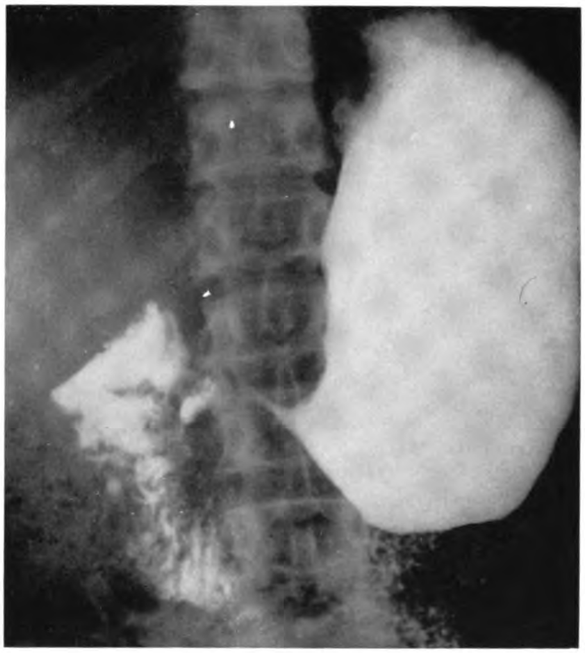

FIG. 195.—Gastric syphilis in a 52-year-old male with upper abdominal pain, belching, vomiting, weight loss and hematemesis. Pyloric deformity due to syphilitic infiltration, verified at necropsy.

remarked that the radiologic appearance of extensive gastric syphilis could not be correlated with the operative findings, where relatively little alteration of the stomach wall was noted. Except in cases of linitis plastica, the syphilitic gastric walls remained relatively pliable. The concurrence of syphilis and carcinoma rendered a diagnosis of syphilis hazardous without histologic confirmation. Others have suggested that the criteria for gastric syphilis include absence of specific treatment, gastric defects, persistence of symptoms on symptomatic treatment but improvement with antisyphilitic therapy, and disappearance of the defect with treatment.

Syphilis of the small intestine occurs infrequently, descriptions being found mainly in older texts. The changes are mainly multiple gummatous plaques which disintegrate into encircling and stenosing ulcers resembling those of tuberculosis. No case reports of the radiologic manifestations of this condition are available. In 1932 we followed a 17-year-old boy admitted because of intestinal obstruction, with characteristic changes on scout abdominal films. He had had marked weight loss over a short time, and vomiting had been present for 2 weeks. The abdomen was distended and visible peristalsis was evident. Abdominal paracentesis yielded 2 liters of clear fluid. At necropsy a constriction about 2 cm. wide encircled the bowel, just above the ileocecal valve with a similar band about 35 cm. proximal to the first. The bowel was markedly dilated. On opening it the mucosa distal to the first constriction was normal, but at the proximal constriction the gut was markedly thickened. The mucosa contained numerous superficial ulcerations which communicated with one another by tunnel-like formations. Microscopic examination of scrapings from the ulcers revealed spirochetes. Studies for other organisms were negative.

Syphilis of the colon is uncommon. In a case reported by Bassoe multiple ulcers were seen occurring as smooth ring-shaped tumors or infiltrations like linitis plastica. Gummatous infiltration also occurs in the mesentery. Syphilitic strictures of the colon are characterized by a stiff canal with smooth walls, and without clear cut demarcation from the normal intestine. No specific symptoms are present. Tuberculosis may produce a similar change, but occurs more often in the cecum and ascending colon.

Rectal syphilis with extensive soft tissue change once was well known as a disease seen mainly in females. Tissue destruction was extensive, with multiple fistulas leading to the perineum, vagina, bladder and the abdominal cavity.

Syphilitic involvement of the spleen is not uncommon in the congenital form of the disease, and is manifested by splenomegaly in newborns. Gummatous changes in the liver once were very common, but rarely are seen today. The pancreas is involved by gummas either as an infiltrating lesion or as a mass simulating tumor of the head of the pancreas, as occurred in a case reported by Castigliano.

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REGIONAL ENTERITIS

Regional enteritis is a disease of unknown etiology, originating as an acute inflammation of the submucosal and subserosal lymphatics, progressing to a chronic granulomatous form. The disease occurs at any age from infancy on, but usually appears in early adult life. Howard, Pulaski and Morrissey reported 2 children 2½ and 5½ years old in whom the clinical picture was that of acute appendicitis, with intestinal obstruction in the first and a more chronic course because of jejunal involvement in the second. These authors noted that in children the clinical picture is one of lower abdominal pain which subsides spontaneously. The diagnosis of regional enteritis should be considered when the pain is unexplainable. The pathologic changes include thickening of the bowel wall, with mild edematous changes, marked enlargement of the mesenteric and intra-abdominal lymph nodes, free fluid, and sometimes later adhesions and perforations. As a rule the terminal ileum and adjacent colon are the primary sites in children. Relatively few cases involving other areas have been reported. Some patients with childhood regional enteritis have been followed for as long as 13 years.

Starting with the basic contribution of Crohn, Ginzberg and Oppenheimer in 1932, the chief site of occurrence appeared to be the terminal ileum. However, it soon became apparent that the disease was widespread. While most common in the ileum, the jejunum, duodenum and rarely the stomach also were affected. The question as to extension into the colon has come in for considerable discussion, and some believe that similar granulomatous lesions of the ascending colon is part of the same disease. The ulcerating exudative inflammatory lesion of ulcerative colitis is quite different from that of regional ileitis. Segmental colitis, or right-sided ileocolitis, may represent a pathologic entity intermediate between these two processes.

In order to understand the clinical picture of regional enteritis, the variability of the pathologic changes and of symptoms must be kept in mind, together with the understanding that no specific relationship insofar as symptoms is concerned necessarily is present. Often the clinical picture of the early stage is explainable on the basis of the acute inflammatory serosal changes.

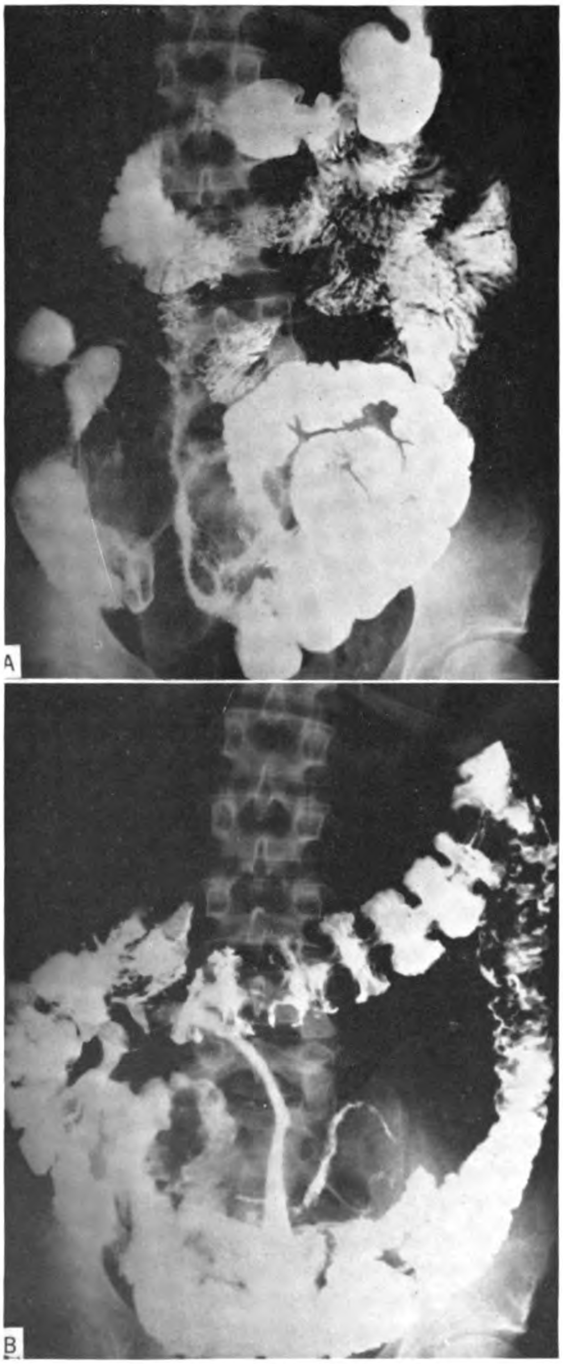

FIG. 196.—*A*, Diffuse regional enteritis involving ileum in a 19-year-old boy. Three-hour film shows dilatation of jejunum and narrowed, fixed loops of ileum.

B, Same patient, 5-hour film. The ileitis presents the same pattern of narrowing and fixation indicative of a classical string sign, together with spastic changes in other loops.

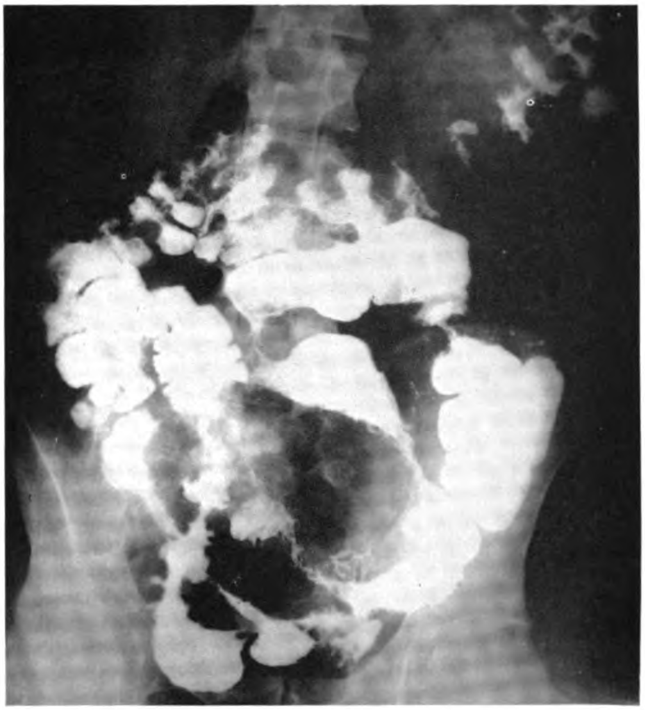

FIG. 197.—Jejunoileitis in a 25-year-old male with lower abdominal pain and diarrhea for 10 months.

Thereafter, however, a wide-spread disparity often exists between symptoms and the anatomic effects of the granulomatous changes. Early in the disease acute appendicitis is simulated so often that the diagnosis often is followed by operation. This must be guarded against whenever possible, because appendectomy under such circumstances may be followed by fistula formation. At such operations it has been possible to observe the appearance of the bowel carefully. The appendix is normal, but the serosa of the ileum is edematous and reddened, with thickening of the mesentery and swelling of the regional lymph nodes.

Roentgenographic examinations of the lesser bowel made a short time before operation in 4 such cases, and soon after operation in 2 others, revealed diffuse ileal dilatation with variable thickening of the mucosal folds and occasional segmentation. A tendency towards hypomobility was observed. These changes could not be considered specific, in striking variance with the characteristic changes seen later in the disease.

With progression of the process, proliferation of the submucosal and subserosal lymphatic epithelium results in lymphatic obstruction. This produces a considerable edema of the bowel walls, particularly of the subserosal and submucosal coats. The major microscopic changes were well described by Otani. First there is a granulomatous enteritis, with thickening of the wall and acute inflammation, sometimes with submucosal abscess formation. Later, the granulomatous changes are more pronounced and the acute inflammation subsides. Focal collections of plasma cells and lymphocytes are found in the thickened, fibrotic submucosa. Otani regarded the advanced granulomatous enteritis with acute inflammatory changes as the predominant lesion. As the disease progresses the involved lesser bowel slowly becomes a thick, rigid, segmented loop with intervening skip areas. The question as to involvement of skip areas can be settled only by microscopic study of excised pieces of bowel because histologic changes can exist in what appears to be uninvolved small

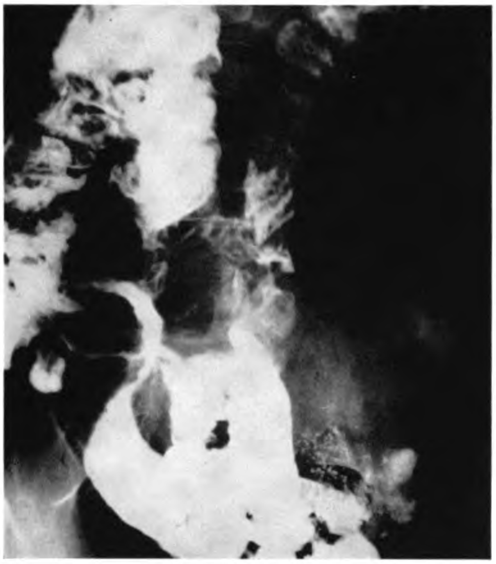

FIG. 198.—Regional enteritis involving terminal ileum with ileocolic fistula to cecum and hepatic flexure, and ileo-ileal fistulas. Verified at operation. Patient is a 30-year-old male with abdominal pain, distention, loss of 40 pounds and history of nausea, vomiting and diarrhea for 5 years.

intestine. The distances between the obviously involved bowel and the apparently normal skip areas vary considerably.

Microscopic study of the advanced lesions of regional enteritis discloses multiple small nodules containing scattered giant cells in the submucosa, the muscularis and serosa. These extend into the perintestinal fat. Central areas of necrosis are seen in these tubercle-like structures. Mucosal ulcers separated by areas of granular mucous membrane appear in the involved bowel when the primary granulomatous process is overshadowed by acute and chronic inflammation. An acute purulent exudate is present over these ulcers. Late in the disease the entire process is obscured by submucosal and serosal fibrosis and scarring. In this stage the loops may be markedly narrowed and relatively fixed in position, with thickened walls and nodular mucous membrane. Varying degrees of partial obstruction are produced by these changes.

The clinical picture of regional enteritis is variable. Some patients have severe distress, and in others the disease goes practically unnoticed for long periods. The history often dates back for a considerable time before the patient comes to his physician. Intermittent abdominal pain and anorexia is accompanied by loose stools, sometimes with mucous or blood. Abdominal distention, weight loss and constipation also have been observed. The symptoms suggest intestinal obstruction or an acute inflammatory change, and a decision as to whether or not to operate often is difficult. In patients with a more advanced enteritis a mass sometimes is present, particularly when serosal extension and matting together of the intestinal loops has occurred. In elderly patients malignant disease may be suspected because of a palpable mass and the gastrointestinal complaints.

Regional enteritis affecting the stomach is uncommon, and usually is seen together with disease of the lesser bowel. However, it may be present when the small intestinal involvement is relatively mild. In a report of 2 cases involving the stomach, Martin and Carr observed that a sharp demarcation was present between the pathologic and normal segments, with absence of the antral mucosal folds and those of the upper duodenum. Below this the mucosal folds were normal. Both cases were verified histologically. Pyloric obstruction and duodenal deformity also were mentioned by Richman, Zeifer, Winkelstein, Kirschner and Steinhardt. In their third case, a 10-year-old boy, diarrhea and abdominal pain had been present for 4 years. A gastrointestinal examination 2 years before revealed narrowing and rigidity of the prepyloric area and a deficiency pattern of the small bowel. At operation the pylorus was involved in an inflammatory mass, and the entire small intestine was diffusely inflamed. No biopsy was obtained, but it appeared that this was nonspecific granulomatous inflammation of the stomach and lesser bowel. The pathologic changes in the stomach in verified cases include thickening and rigidity of the walls, engorgement and reddening of the serosa and diffuse enlargement of the lymph nodes. When acutely inflamed the stomach walls may become sufficiently thickened to simulate neoplasm.

Duodenal involvement with regional enteritis is somewhat more frequent. McGarity

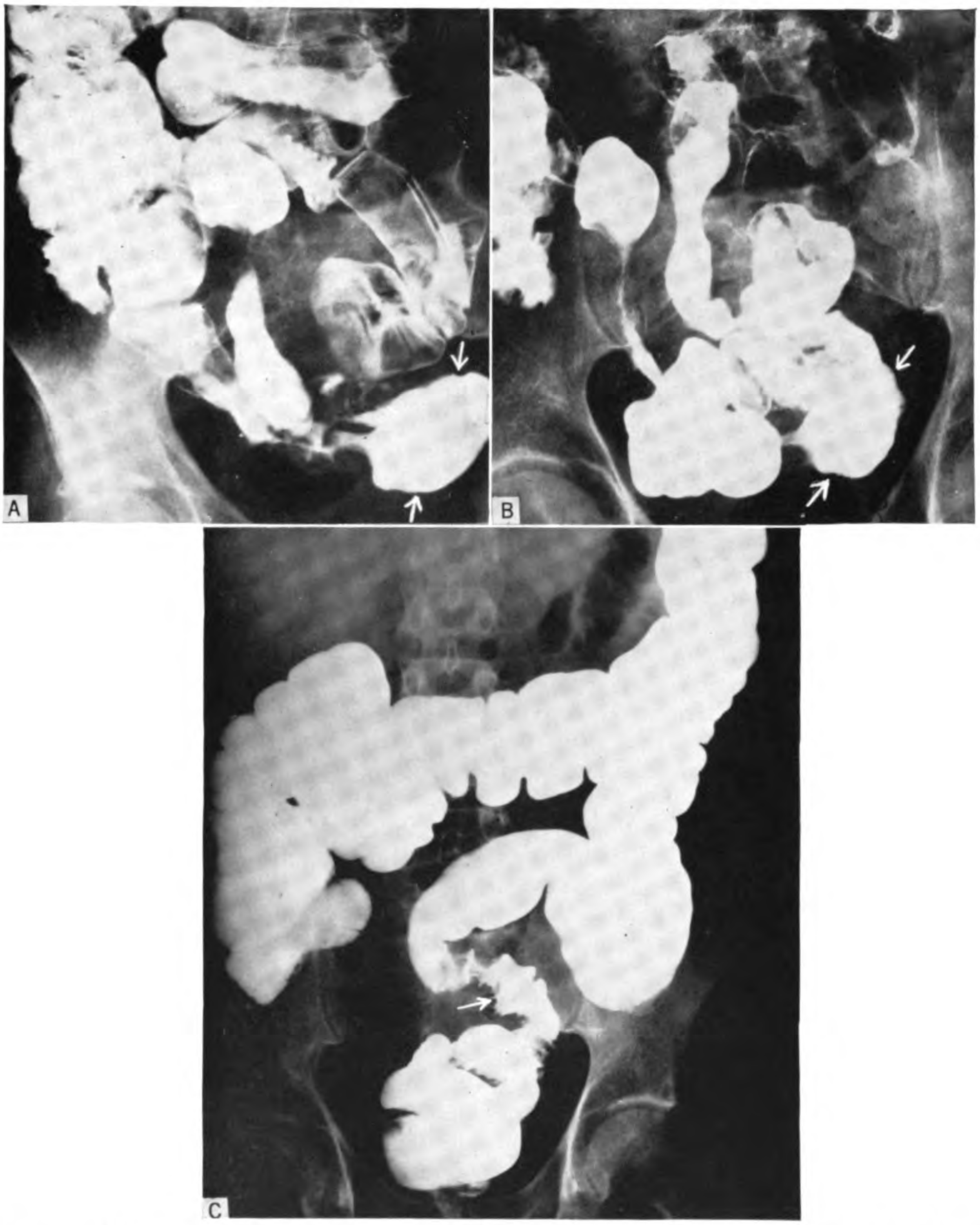

FIG. 199.—*A*, Cone-down study of right lower quadrant of a 48-year-old man with persistent right lower abdominal pain, weight loss, intermittent diarrhea A distended fixed loop of ileum is seen (arrows) in the lower pelvis to the right of the midline.

B, Same patient, about $\frac{1}{2}$ hour later. Note change in appearance of ileum, which now assumes a string-like appearance. The fixed loop has not altered its position (arrows).

C, Same patient, barium enema revealing irregular indentation in sigmoid (arrows). At operation this was due to fixation of acutely inflamed ileum against the serosa of the sigmoid. Microscopic examination indicated acute serosal involvement with regional enteritis.

gathered 25 cases from the literature, and added another. While this may exist as an isolated lesion at first, it later develops into a generalized form. Radiologically, duodenal enteritis is accompanied by disturbed mobility and deformities with narrowing which ultimately may become obstructive with consequent gastric dilatation. The duodenal mucosa becomes irregularly granular, with sudden or gradual transitional areas. With further progression fistulous tracts may appear.

As a rule regional enteritis of the ileum and to a lesser degree, the jejunum, is the common pattern. There are cases in which the jejunum is primarily affected, although these extend into the ileum and duodenum eventually. Three proven cases of jejunal enteritis, two of which also had duodenal extension, were mentioned by Janus. He noted that the greater curvature of the stomach was spastic and irritable because of the adjacent inflammation of the duodenum or jejunum.

The radiologic diagnosis of regional enteritis depends on observation of changes apparent in the passage of barium, the demonstration of abnormal mucosal patterns, the identification of stenotic and fixed loops and of fistulous tracts. It is not possible to estimate the patient's condition purely on the basis of radiologic change. Patients with radiologic pictures of extreme involvement may have absent or mild symptoms. On the other hand, severe complaints often accompany relatively minor radiologic changes.

Lesser bowel changes are best identified on gastrointestinal examination. Visualization of the terminal ileum by barium enema also may offer evidence of ileitis by revealing a narrowed fixed loop. Whenever the diagnosis is suspected because of changes seen on barium enema, investigation should be continued by gastrointestinal examination with special reference to the small intestine. Lesser bowel dilatation is frequently observed with regional enteritis, but this may be intermittent and variable, particularly in the early stage. Granular mucosal markings, obliteration of folds, hypermotility and hypomotility, segmentation and areas of stenosis all are present in different degree during the various stages of the disease. None serve to elucidate clearly the wide variety of pathologic patterns existent with this condition. In the stage before actual rigidity of the intestinal loops appears, the pre-stenotic stage, a transient and variable pattern of diffuse bowel distention often is present. This may be associated with areas of spasm of sufficient magnitude and duration to simulate an actual stenosis. However, if this is observed by use of the fluoroscope over some time, and if serial films are made, it will be found that a capacity to relax may still exist, and what appeared to be a fixed loop at one moment may be definitely dilated a half hour later. Hence, one must be careful before assuming that a narrowed loop represents an actual stenosis. This diagnosis should not be made unless the loop is fixed and retains its configuration and caliber sufficiently long to convince the observer that it cannot distend. The appearance of the intestine at either end of areas of stenosis or dilatation is inconstant. In some the cut-off between the spastic and non-spastic areas is rather abrupt, while in others it is tapering.

With progression of the disease to the stenotic stage, the lesser bowel loops become fixed and somewhat tubular. The edges of the mucosa in the affected segments are irregular and granular. In some the mucosal pattern is smoothened. These are not necessarily obstructive lesions. If the bowel wall is edematous transient episodes of fairly severe partial obstruction often are observed. The transition between stenotic loops and those not similarly involved may be gradual or quite abrupt.

Fistula formation is a relatively late manifestation of regional enteritis. Among the more common tracts are those between ileal loops, giving rise to ileo-ileal connections. Sometimes a loop of bowel actually is excluded by such fistulas. Retroperitoneal tracts may involve the ureters or the bladder. Involvement of the vagina and uterus has also been described. Infrequently, fistulas to the duodenum or the colon may appear. Tracts leading to the anterior abdominal wall also occur, more often after operations for supposed acute appendicitis.

If a sufficient area of small intestinal mucosa is diseased, the patient develops a

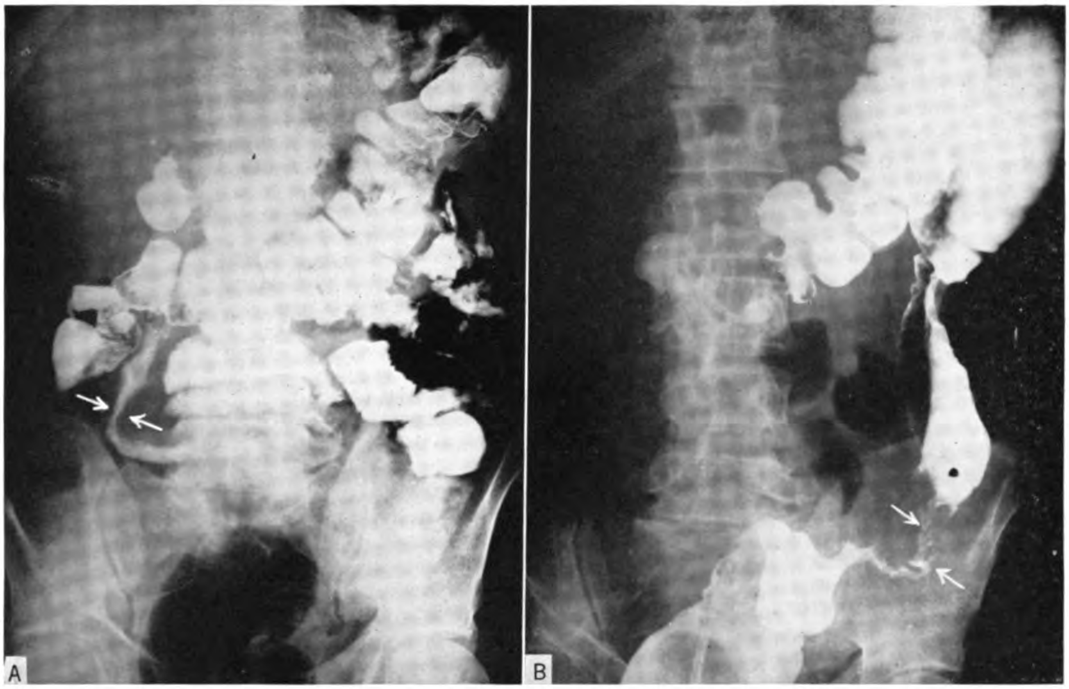

FIG. 200.—*A*, Recurrent regional enteritis in a 47-year-old man operated upon 3 years before. Ileal loop leading into transverse colon at ileotransversostomy presents a string sign (arrows).

B, Same patient 14 months later. Recurrent pain, lower abdominal pain and diarrhea. Barium enema discloses an infiltrating sigmoid lesion (arrows). Operation revealed a stenosing benign granulomatous mass due to perisigmoidal extension from recurrent regional enteritis.

malabsorption pattern with ileus, hypoproteinemia, ascites and other symptoms.

Perforation of the bowel with peritonitis is an unusual complication of regional enteritis. We observed a case in which peritonitis followed perforation of an excluded loop of ileum proximal to a point of stenosis. This loop had been bypassed by an ileoileal fistula.

The question as to colonic involvement with regional enteritis is an involved one. Radiologically, spasm of the cecum and ascending colon is observed fairly often. In a review of 50 of our cases, 16 showed signs of marked spasm and irritability of the cecum and ascending colon. Fixed deformities secondary to pericecal and pericolonic infiltration, part of the exudative stage of serosal involvement was seen in 12 cases. Occasionally the cecum was indented by a mass of granulomatous ileum, and in elderly patients this sometimes suggested a malignancy. In 3 cases fixed loops of lesser bowel against the transverse colon produced radiologic evidence of extrinsic pressure.

The differential diagnostic difficulties between early acute regional enteritis and appendicitis have already been mentioned. In later stages other granulomatous lesions become more difficult to eliminate. With multiple fistulas tuberculosis and actinomycosis have to be considered. Lymphosarcoma may appear quite similar to regional enteritis, although the segmentation and dilatation tend to be more pronounced, simulating the malabsorption pattern of sprue, which likewise may appear with advanced enteritis. Differentiation from Whipple's disease radiologically also is sometimes difficult, but consideration of the clinical aspects helps here.

A rare condition simulating regional enteritis was reported by Zeek and Herrman in a 20-year-old patient. At necropsy this patient had a hypertrophic ileal stenosis due to hyperplasia and hypertrophy of the smooth muscle of the intestine which was neither inflammatory nor neoplastic. This was accompanied by changes in the myenteric nerve plexus. The patient had two siblings with congenital pyloric stenosis and a third with Hirschprung's disease. The lesion was considered probably of congenital origin, but possibly intensified during puberty by unknown factors influencing the autonomic nervous system.

The differential diagnosis between ulcerative colitis with terminal ileal involvement is not difficult if one recalls that back-wash ileitis is relatively limited in extent, and often is seen with pronounced changes of the entire colon. More difficulty may be encountered with the differential diagnosis between segmental or right-sided colitis and regional enteritis.

The rate of recurrence of regional enteritis is appreciable. One of the chief sites for further progress is at the enterocolostomy stoma in postoperative patients. These individuals sometimes complain of considerable pain, and radiologic investigation may fail to reveal sufficient evidence to support the diagnosis of recurrence at the stoma. Careful investigation of the operative site for irritability and spasm, together with meticulous examination of the mucosal patterns may help identify some alteration to support the diagnosis. With more advanced lesions, stiffening of the bowel wall, smoothening of the mucosa and fixation of the loops appear.

Malignancy as a complication of regional enteritis is rare. Ginzburg, Schneider, Dreizin and Levinson reported a 30-year-old male patient with carcinoma arising in a segment of bowel with granulomatous jejunitis.

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SARCOID

Sarcoidosis is a disease of unknown etiology which produces granulomatous lesions superficially resembling tuberculosis in many tissues, principally the skin and lymph nodes. The lungs, mediastinum and bones also are affected, and hepatosplenomegaly may be quite prominent. If nothing specific is noted radiologically, the diagnosis may have to be established by biopsy.

Involvement of the gastrointestinal tract by Boeck's sarcoid is quite uncommon. Lorber, Shay and Woloshin failed to find any verified cases in their review of the literature. Others have noted changes in the stomach which mimic carcinoma or ulcer. Sirak's case of gastric sarcoid simulated linitis plastica. There apparently was remission of complaints on cortisone treatment, but the radiologic changes were not reversed. Sirak gathered 12 similar cases from the literature. Sarcoidosis causing a persistent pyloric narrowing was described by Orie, Van Ridsell and Van der Zwaag. Occasionally the lesser bowel is affected, simulating regional enteritis. Indeed, before regional enteritis was recognized, it was not uncommon to mistake these cases for tuberculosis or sarcoidosis.

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EOSINOPHILIC GRANULOMA

Eosinophilic granuloma of the upper gastrointestinal tract is considered as a granulomatous disease with an allergic background. This is supported by the presence of peripheral eosinophilia with local eosinophilic gastrointestinal tissue infiltration. The disease occurs as a local, poorly circumscribed submucosal polypoid mass which may become pedunculated and protrude into the antrum, sometimes extending into the duodenum. Concomitant lesions are found in the duodenum and small intestine. Another form is eosinophilic infiltration of the stomach wall which may produce obstruction because of thickening of the pyloric ring and edema of the muscularis. Such a case was described by Spencer, Comfort and Dahlin in a 40-year-old male with recurrent cramps and diarrhea for 11 years. This patient had a blood eosinophilia of 63 per cent, and sternal puncture disclosed a marrow eosinophilia of approximately 50 per cent. Hypertrophy of the pylorus with massive eosinophilic infiltration also gives rise to symptoms and radiologic changes simulating malignancy. The diagnosis cannot be made without histologic examination of the involved tissue. The extent of the eosinophilic infiltrations varies from local to massive lesions involving the entire stomach. The condition is not related to the osseous disturbance with the same name.

The symptoms of gastric eosinophilic infiltration or polypoid granuloma are not characteristic. Recurring abdominal pain, vomiting and symptoms of pyloric obstruction occur with infiltrating lesions. The polypoid form is not associated with specific disturbances, and often is found incidentally. Rigler, Blank and Hebbel, in a report on 9 patients from 37 to 83 years old, found only 4 with symptoms. Two had complaints like those of duodenal ulcer, and pernicious anemia was present in the other two. These authors noted radiologic evidence of shallow ulcerations, particularly at the apex of the polypoid lesions, in 2 patients. Two also had malignant disease, but no association was presumed. Hemorrhage from a polypoid granuloma was a presenting symptom in a 54-year-old woman reported by Smith. At operation this patient had a sessile eosinophilic granuloma in the antrum together with a pedunculated polypoid adenoma.

The size of eosinophilic granulomas is

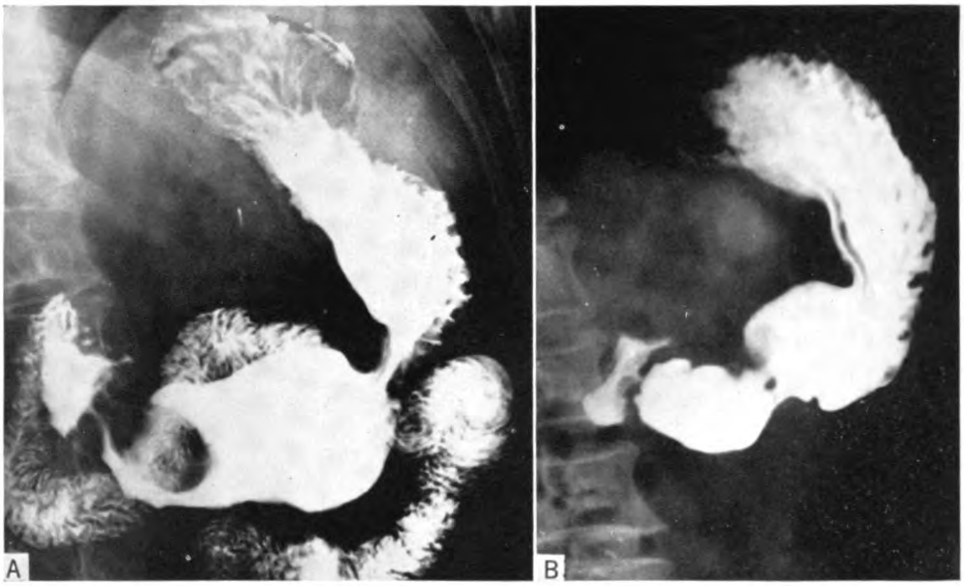

Fig. 201.—*A*, Polypoid eosinophilic granuloma in pylorus. Verified. *B*, Infiltrative pyloric eosinophilic granuloma. Verified.

variable. Some are but a centimeter or two in diameter, and others exceed 5 cm. Of the 2 cases seen by us, one had a large pedunculated polypoid mass which projected into the duodenum, and the other had infiltration of the distal portion of the lesser curvature simulating carcinoma. The second patient was admitted with brisk bleeding.

Infrequently eosinophilic granuloma involves the lesser bowel alone, as reported by Polayes and Krieger in a patient with an exophytic fungating granuloma confined to a single segment of the jejunum.

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NONSPECIFIC GRANULOMAS

Occasionally a granuloma in the gastrointestinal tract has no specific etiology. Some may be due to trauma or perforation associated either with foreign bodies or surgery. We have seen 3 perforating granulomas of the colon which were believed to be carcinomas on *x*-ray examination. In one of these the presenting symptoms were those of a left subphrenic abscess. In another 76-year-old man acute intestinal obstruction appeared soon after prostatectomy. Gastrointestinal examination disclosed a partial block in the midjejunum, with dilatation of the proximal bowel. At operation a granulomatous lesion was found extending from the mesentery to the wall, but not to the mucosa, of the jejunum. This loop was resected and the patient recovered. Microscopic examination of the excised specimen disclosed a nonspecific granuloma.

Granulomas of the cecum associated with perforating foreign bodies and infection about a partially excised stump of appendix were reported by Mincks, Pulaski and Beatty.

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COLLAGEN DISEASES

The collagen diseases include a number of conditions marked by connective tissue disturbances, particularly of the intercellular components, with lesions of the lesser blood vessels and the vasa vasorum of the larger arteries. The diseases are characterized by multiple focal degenerative lesions which weaken the walls of the involved vessels, often producing small aneurysmal dilatations. These vessels also become thrombosed, causing local ischemia. Inasmuch as the disease involves vessels anywhere in the body, a tremendous diversity of symptoms and pathologic changes are produced. Included among the collage diseases are polyarteritis nodosa, disseminated lupus erythematosus, dermatomyositis and scleroderma. Diseases such as rheumatic fever and rheumatoid arthritis also are believed based on collagen disturbances. Wegener's granuloma is regarded as another manifestation of this process, presenting with granulomatous ulcerations of the face, pulmonary lesions, and in common with the other collagen diseases, widespread necrosing arteritis. Whipple's disease likewise may have a common underlying mechanism.

Involvement of the gastrointestinal tract with *polyarteritis nodosa* is not uncommon, but symptoms are mild or absent. Abdominal pains are present even without detectable lesions. Occasionally pain is a precursor of an acute disturbance, possibly perforation of the bowel. If the patient has been on antibiotic or steroid therapy the possibility of ileus and perforation may be increased. The interference with the blood supply to the gastrointestinal tract may cause infarcted areas, followed by ulceration and hemorrhage. The pain sometimes simulates acute appendicitis or other ulcerating lesions. With perforation and peritonitis the acute picture of shock predominates. The radiologic diagnosis is not specific. In a review of 28 cases, Garland and Sisson found no abnormality other than a pattern of ileus.

Gastrointestinal involvement with *scleroderma* is seen fairly often. The radiologic picture of esophageal atony and dilatation with diminution or absence of peristalsis is well known. As with other collagen diseases, the vascular involvement results in atrophy, cellular infiltration and fibrosis of the muscular structures. In a review of the literature Abrams, Carnes and Eaton found 21 cases of disseminated scleroderma with sufficient *x*-ray or pathologic data for analysis, and presented 6 additional cases. They noted that intestinal involvement with scleroderma sometimes has a rapidly progressive fatal course. No correlation was found between the cutaneous disturbances or skeletal disability and the gastrointestinal changes. Ileus was the predominant manifestation, with poor peristaltic activity and persistent bowel distention. The picture, in their opinion, simulates that seen in the esophagus. Occasionally gastrointestinal scleroderma results in sufficient ileus to mimic intestinal obstruction, as occurred in the case reported by Arcilla, Bandler, Farber and Olivar. In this patient operation disclosed enormous distention of the entire small intestine and the colon to the rectosigmoid.

Death from paralytic ileus secondary to generalized scleroderma was reported by Prowse. With less pronounced neuromuscular disturbances, delayed motility and dilatation of the bowel results in a picture similar to that of the malabsorption pattern.

Scleroderma of the colon is infrequent. In the 2 cases reported by Hale and Schatzki questionable involvement was indicated by a peculiar sacculation of the bowel, with

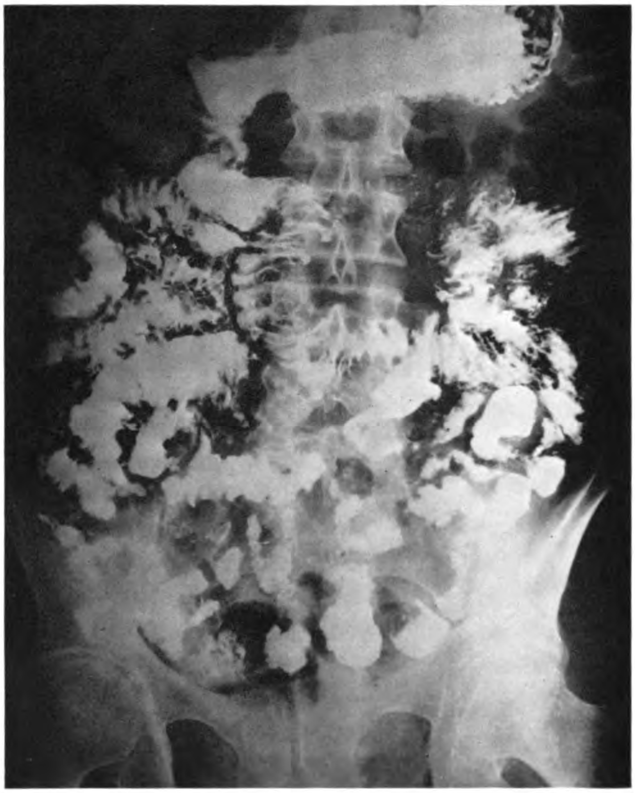

FIG. 202.—Verified Whipple's disease (intestinal lipodystrophy) in a 54-year-old man. Note the widened valvulæ conniventes, segmentation and scattering of barium in small intestine.

increased rigidity between the areas of sacculation in 1 case.

Lupus erythematosus is more common in young women, and is accompanied by an erythematous facial rash together with disseminated cutaneous lesions, and proliferation and inflammation of the serous linings and endocardium. The arteriolar and arterial disturbances are widespread, with a striking tendency for involvement of the endocardium and pericardium. Gastrointestinal involvement with systemic lupus is infrequent. In a review of 87 cases Brown, Shirey and Haserick found 55 with no abdominal symptoms and 25 with minor complaints. Seven had severe paralytic ileus. This was segmental in 5, affecting the stomach in 1 patient, the duodenum in 3 and the jeunum in 1. All 7 patients had been on steroid treatment. The symptoms were ascribed to vascular disturbances of the bowel wall. Two patients also had ulcerative colitis. Necropsy was obtained in 11 of the 87 patients and no gastrointestinal changes were observed in 9. Interstitial edema and submucosal hemorrhage on the small intestine was found in 1, and collage deposition was noted in the submucosa of the stomach in another. Necropsy disclosed no cause for the ileus in 5.

The uncommon combination of lethal granulomatous midline facial ulceration known as malignant granuloma or chronic infectious granuloma is referred to as *Wegener's granuloma* when associated with polyarteritis. A case with pronounced jejunal changes due to necrotizing arteritis was reported by Geist and Mullen in a 31-year-old male who complained of intense nausea. Scout abdominal films showed multiple dilated loops of jejunum similar to those seen with sprue. At necropsy an acute necrotizing arteritis involving the kidneys, muscles, lungs, esophagus and small intestine was found, together with nonspecific chronic inflammation of the upper respiratory passages.

Whipple's disease, or intestinal lipodystrophy, is characterized by progressive loss of weight and strength, indefinite abdominal disturbances, migratory polyarthritis with pain, swelling, and increased heat like that of rheumatoid arthritis. Involvement of the serous membranes causes persistent pleurisy, pericarditis and peritoneal irritation. The lesions, which basically deposit fat and glycoproteins in the intestinal and mesenteric lymphatic tissues, also may involve the mesenteric and retinal vessels. The stools consist chiefly of fat and fatty acids. The arthritis may disappear during attacks of diarrhea, and later in the disease the clinical picture simulates sprue. A diffuse lymphadenopathy pigmentation and fever may be present, with microcytic anemia and low serum proteins. The pathologic changes encountered include macrophages in the walls of the small intestine and fatty granulomatous changes in the mesenteric lymph nodes. There is thickening of the villi of the small bowel, and the mucosa becomes shaggy and thickened.

The radiologic changes present with Whipple's disease are not specific, and reflect the malabsorption pattern with distention, segmentation, hypomobility and mucosal abnormalities. In a case reported by Eyler and Doub, there was widening of the duodenal loop due to a mass of retroperitoneal lymph nodes.

The *Grönblad-Strandberg syndrome* is a hereditary condition characterized by cutaneous, ocular and visceral manifestations due to a widespread disorder of the elastic tissues. Degeneration of these tissues is accompanied by angioid retinal streaks. The cutaneous disorders are bilateral and symmetrical. The neck, flexural folds of the axilla, elbows, knees and groin are affected. The skin is soft, inelastic and papules vary from a few millimeters to about a centimeter in diameter are noted under the cutaneous lines. The disease may be present in infancy, and often goes unnoticed. Biopsy reveals marked alteration of the elastic tissue in the central and deep parts of the skin with frayed, coiled fibers. Calcification may occur in the involved tissues. These patients may present with massive hemorrhage. Three such instances during pregnancy were reported recently by McCaughey, Alexander and Morrish. The diagnosis sometimes is suggested by an acquaintance with the clinical aspects of the disease. No radiologic change is associated with this disorder.

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METABOLIC DISTURBANCES

Malabsorption Disorders.—Poor absorption from the gastrointestinal tract is manifested by the diverse effects of disturbed water metabolism and deficient absorption of fats, proteins, carbohydrates, minerals, vitamins and electrolytes. Primary malabsorption is seen as celiac disease in infancy and childhood, and as sprue in adults. These are considered the same disease. The process occurs in a relatively acute form, or is insidious in onset. A deceptive clinical picture is present when a mild or incomplete form of the malabsorption syndrome is present. The disease is genetically transmitted, and has a familial tendency. With primary sprue, no pathologic changes are visible on gastrointestinal examination. The condition may go undiagnosed in its incomplete form. In the advanced form steatorrhea accompanied by anemia, weakness, anorexia, weight loss, pallor and abdominal distention often is pronounced. Edema and ascites reflect the hypoproteinemia, and hypocalcemia may be manifested by osteomalacia and tetany. A wide variety of changes due to hypovitaminosis, hyponatremia and hypokalkemia also may be present. Hepatic disorders with jaundice, skin pigmentation, glossitis and stomatitis are observed. The stool of sprue is characteristically foamy, bulky, light colored and contains much undigested fat. However, it is possible even in fairly well-advanced cases to observe nothing more than light, bulky formed stools, which on chemical analysis have an abnormal fat content.

Celiac disease in infancy is accompanied by malnutrition, marked abdominal distention and a doughy soft abdomen with thin musculature. The limbs, buttocks and groins are wasted, the skin becoming quite wrinkled. Gross retardation often is present. Clubbed fingers also occur, but disappear as the condition comes under control. The stool varies from a large stool with undigested food to frothy, fatty foul stools. Either diarrhea or constipation may be present, but diarrhea is more common.

Considerable help in diagnosis can be obtained from laboratory examinations, particularly the identification of a flat glucose tolerance and vitamin A absorption curve, and the demonstration of normal pancreatic enzymes. Study of the blood serum for protein, calcium and phosphorus content sometimes help. Examination of the stool for fat is useful when steatorrhea is not prominent. Recently technics have been advanced for the procuring of intestinal biopsies. Mucosal atrophy confined mainly but not entirely to the villi has been observed in celiac disease by Sakula and Shiner, much the same as observed in sprue in adults.

The radiologic identification of celiac disease in infants is not difficult. Unusual protrusion of the abdomen is manifest on plain films, together with rather pronounced distention of the lesser and large bowel. Mixed gas and watery material in the small intestine produces a bubbly appearance, and fluid levels form in some loops. The transit time of barium is slow, with a tendency towards segmentation and flocculation. In later stages of celiac disease motility is delayed further, with pronounced segmentation and flocculation in loops of dilated bowel. In some the borders are smooth, and in other segments prominent valvulæ conniventes are visible. When the bowel is partly filled, it may appear rather granular because of the mixture of mucous and barium. The appearance of the loops vary from moment to moment. Distention of the colon usually is less prominent than small intestinal dilatation, but occasionally pronounced atonicity and dilatation of the colon is present.

The radiologic changes in sprue likewise are quite variable in the milder stages. Nevertheless, the same pattern of slow passage and distention of lesser bowel is present. The jejunal and proximal ileal loops tend to be more dilated than the terminal ileum. The smoothening of the bowel wall in some loops, and the presence of prominent valvulæ

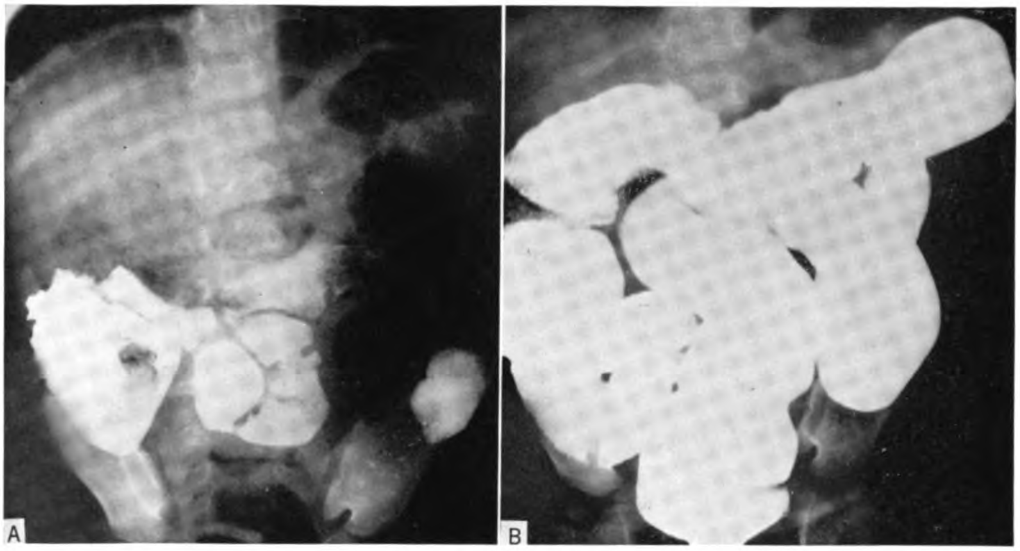


FIG. 203.—*A*, Celiac disease in a 17-month-old female child. Small intestinal examination shows dilatation of intestinal loops, segmentation and smoothening.
B, Barium enema, same patient, reveals an atonic, dilated and redundant colon.

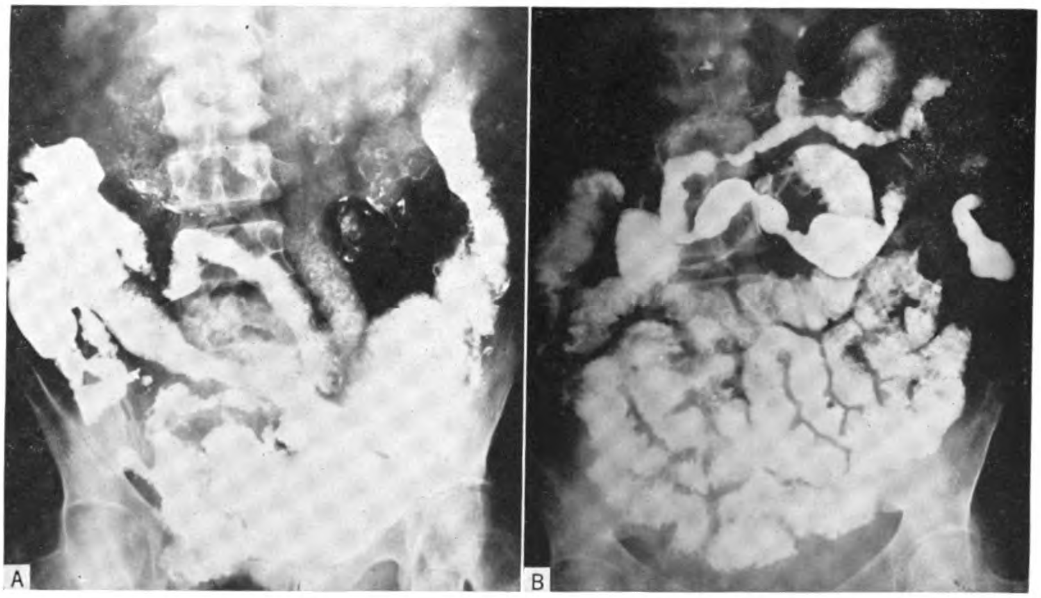
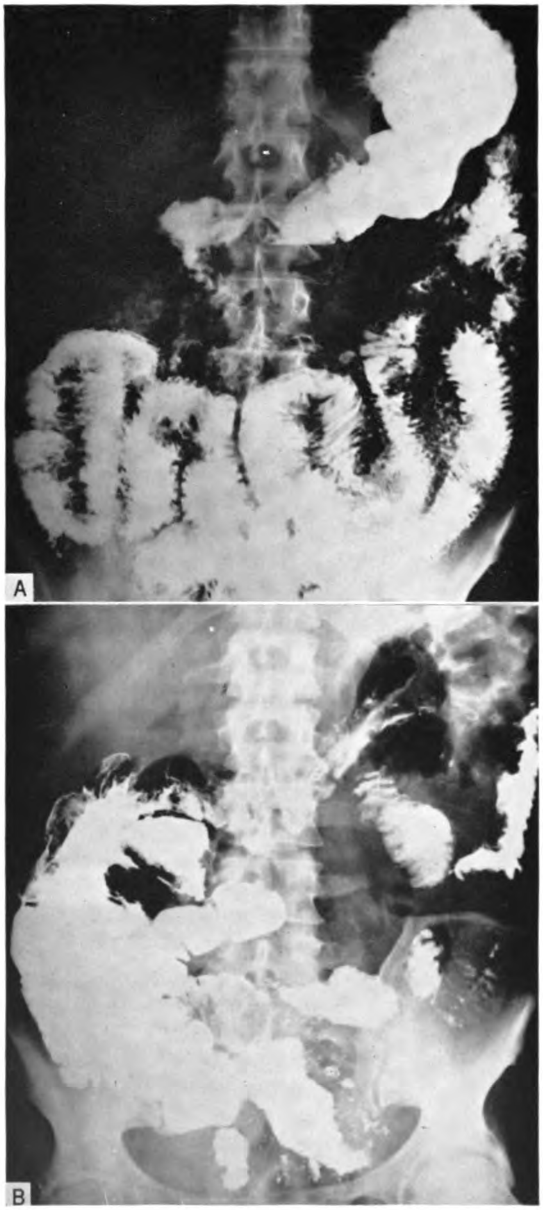


FIG. 204.—*A*, Small intestinal study in a 48-year-old woman with sprue. Three-hour film shows flocculation of barium, slow transit time, smoothened bowel walls and tendency to segmentation.
B, Same patient, 4-hour film.



205.—*A*, Sprue in a 52-year-old woman, known for more than 20 years, with watery, foul, greasy, frothy stools. Small intestinal study shows feathery pattern of small intestinal mucosa.
B, Same patient, 2 hours later. Segmentation and smoothening of lesser bowel pattern.

conniventes in others, is rather common. The term "moulage sign" is applied to relatively long, distended loops with smooth borders. Between these are varying lengths of empty or partly inflated loops containing relatively small amounts of barium. The mucosal pattern may be irregular. No fixation of bowel is identified, nor do fistulous tracts form. The colon may be elongated and atonic, with smooth walls. However, on post-evacuation films the mucosal pattern is relatively normal.

Dilatation of the bowel with sprue is more prominent than that associated with secondary malabsorption patterns seen in the later stages of jejunoileitis, small intestinal lymphomas, Whipple's disease, amyloid and collagen diseases. Pancreatogenous steatorrhea due to pancreatitis, carcinoma of the pancreas or with obstruction of the pancreatic ducts produce a similar but less pronounced pattern. Cystic fibrosis in infancy has a pattern of lesser bowel distention similar to that of celiac disease. The concomitant pulmonary changes and other clinical manifestations serves to differentiate these two conditions. Changes in the lesser bowel following vagotomy or the use of blocking agents sometimes simulates a malabsorption pattern.

Occasionally sprue is accompanied by severe osteomalacia. Juergens, Scholz and Wollaeger reported a group of patients in whom sprue was not accompanied by the usual steatorrhea. In 5 the presenting complaints were referable to the musculoskeletal system, while gastrointestinal symptoms were minimal. In 3 deficiency patterns of the small intestines were seen, and in 1 the lesser bowel was normal. No radiographic studies were available in the fifth patient. Prolonged jaundice with steatorrhea likewise may be associated with pronounced skeletal alterations. Atkinson, Nodrin and Sherlock reported 25 patients with prolonged obstructive jaundice, 3 due to hepatocellular jaundice and 22 to intrahepatic or extrahepatic biliary obstruction. Seven had severe osteomalacia and osteoporosis.

Another interesting complication is volvulus of the colon, as reported by Glazer and Adlersberg. They described 9 patients who came to necropsy. Three had intestinal obstruction. A fourth case was recognized and treated successfully. Two had sigmoid volvulus, one cecal volvulus and the fourth had an angulation or volvulus at the splenic flexure. All had been known to have sprue from 7 to 23 years.

The response to treatment of sprue may be gratifying. Some improvement of the gastrointestinal pattern was identified after liver therapy by Hornsby and Baylin. Adlersberg and his colleagues reported that steroid therapy relieved the symptoms, but that clinical improvement was not paralleled by the radiologic changes.

Steatorrhea may follow a subtotal gastrectomy, particularly with extensive removal of stomach. This, too, may be associated with bony changes, as occurred in a case reported by Pyrah and Smith. Steatorrhea has been reported as a complication of longstanding diabetes mellitus with peripheral neuropathy. This infrequent complication is attributed to impairment of the autonomic nervous system.

While it is generally accepted that nontropical and tropical sprue are the same disease, certain differences have been pointed out by Comfort and Wollaeger. They noted that most patients with tropical sprue are undernourished, their diet being particularly insufficient insofar as protein content is concerned, whereas in nontropical sprue dietary intake of protein is adequate. Vitamin D deficiency with osteomalacia reportedly is rare with tropical sprue, but more frequent with the nontropical form. Differences in response to treatment also have been noted.

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Amyloidosis.—Deposition of amyloid occurs in a primary form, or as a complication of chronic disease, particularly inflammations such as empyema or osteomyelitis, or with ulcerative colitis, rheumatoid arthritis, syphilis or subacute bacterial endocarditis. It also has been observed in association with myeloma, lymphoma and leukemia. Amyloid is a homogeneous substance of unknown composition, presumably related to carbohydrates. This material is deposited in the media of the smaller arteries and in the perivascular connective tissues. Because of this generalized vascular predisposition, the clinical picture reflects a widespread pathologic distribution.

Primary amyloidosis is not as frequent as the secondary type, and has a tendency to involve the heart more often than the parenchymatous organs. It is still not clear whether these two disturbances represent the same disease process. The diagnosis of amyloidosis is aided by the determination of Congo red retention. Examination of biopsy material from the gingiva and liver often is helpful.

Amyloidosis follows a chronic course, terminating in failure of the heart, kidneys, liver or adrenals. Progression sometimes is fairly rapid, but the primary is regarded as slower than the secondary variety.

Gastrointestinal involvement with amyloidosis occurs in a relatively limited form, or more frequently as a diffuse infiltration. Gastric involvement as an isolated lesion is rare, occurring more often as part of diffuse gastrointestinal amyloidosis. Amyloid is deposited in the gastric mucosa and in the villi of the small intestine. From this it extends into the muscular and serosal coats. Vascular changes in the stomach and lesser bowel result in rigid, stiffened vessels surrounded by amyloid infiltrations. Involvement of the colon is less frequent, and usually less extensive than seen in the small bowel. The serosa becomes thickened and leathery, presenting a congested appearance with small gray flecks resembling miliary granulomas. In the later stages the entire intestine is reduced to a tubular, and sometimes dilated structure, with the small intestine of greater caliber than the colon. Ulceration of the mucosa is not infrequent, and submucosal hemorrhages may become prominent.

The clinical picture of amyloidosis is complicated, varying from minor gastrointestinal complaints to acute disturbances with hemorrhage, epigastric pain and vomiting, anemia and rapid weight loss suggesting malignant disease. Pyloric obstruction due either to ulceration or diffuse infiltration with amyloid sometimes occurs. Diarrhea also may be a prominent symptom with lesser bowel amyloidosis, and ileus indistinguishable from intestinal obstruction can be confusing.

The radiological aspects of amyloidosis reflects the diversity of the pathologic changes. The gastric lesions can simulate pyloric ulcer, or carcinoma may be suggested when infiltration, rigidity and filling defects of the antrum occur. Sometimes the gastric rugæ become markedly thickened and peristalsis is impaired. However, massive rugal folds without peristaltic disturbance also has been observed. With duodenal involvement a clinical and radiologic change suggestive of peptic ulcer may follow. Amyloidosis of the small intestine first is manifested by a delay in the passage of barium. In some, concomitant dilatation and smoothening of the loops point towards a malabsorption pattern, but as a rule this is not as pronounced as that of sprue, nor does it have the rigidity and tubulation associated with enteritis. The mucosa of the small intestine sometimes is prominent, and in other areas may be quite flattened. Ileus can become so extensive as to be fatal.

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Hemochromatosis.—Hemochromatosis is a chronic disease of unknown origin, probably an inborn metabolic error, in which there is extensive deposition of iron in the body tissues. The skin, particularly over the genitalia, extremities and face becomes bronzed by melanin deposition or blue-gray because of iron pigment deposits. The pigment is deposited intracellularly. Hemosiderin produces a cellular destruction followed by fibrosis. The process occurs most often in the liver, skin and pancreas, producing cirrhosis, skin pigmentation and diabetes. Mucosal deposition of pigment also has been observed. The disease appears in adults, more often in males. Cirrhosis is followed by esophageal varices and ascites. Diabetes may be present before the underlying disease becomes apparent. Weakness and weight loss are prominent, and the patient may complain of severe pain over the liver. Testicular atrophy and impotency appear later.

Radiographic examination is of little help in the diagnosis of hemochromatosis. The enlargement of the liver, and occasionally of the spleen, may be identified on scout abdominal films. Sosman described a zone of radiolucency immediately beneath the right diaphragm which may correspond to a zone of diminished pigment deposition just beneath the capsule of Glisson. Liver biopsy sometimes is required to establish the diagnosis.

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Porphyria.—Porphyria is an anomaly of porphyrin metabolism which may be familial in origin, and is accompanied by remissions and exacerbations of bizarre gastrointestinal and central nervous system disturbances. The condition is considered a reversion to an embryonic type of pigment metabolism with formation of excessive uroporphyrin, believed to be derived from the porphyrins of ingested plant and animal tissue rather than from breakdown of hemoglobin. The disease rarely occurs as a congenital condition apparent in infancy or early childhood. More often it is present in adult women as a relatively acute disorder or as a toxic manifestation secondary to agents such as barbiturates, lead, sulfones and acetanilid which probably act as precipitants in patients with the underlying metabolic disorder. The disease is identified by recognizing the wine-colored pigment in the urine. Occasionally the urine is normal in color when excreted, but the wine color appears after exposure to sunlight. This is due to the fact that the pigment is excreted as porphobilinogen, which is colorless. Porphyria also occurs as a chronic disease in females, often associated with diabetes and liver disease. Photosensitivity may be important in precipitating attacks of pain.

Inasmuch as porphyrin has a widespread depressant effect on cellular metabolism, the pathologic changes may be extensive. There is a direct effect on the innervation of the bowel wall which may cause severe ileus. The pain associated with this distention sometimes simulates an acute surgical condition. Occasionally, mechanical disturbances occur because of the marked relaxation of the bowel. A case of acute porphyria with volvulus and gangrene of the cecum was reported by Watson, Varco and Schmid. More often acute pain with porphyria presents no changes on the gastrointestinal roentgenograms.

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Glycogen Storage Disease.—Glycogen storage disease is the term applied to a group of diseases of infants characterized by abnormally large amounts of glycogen in the tissues. The disease is congenital or familial, and is believed to be associated with defects in the enzymatic aspects of glycogen metabolism which result in increased storage in the liver, kidneys, heart and muscles. There are no specific abdominal radiographic changes other than those which may be ascribed to diffuse enlargement of the liver or kidneys. In patients who develop cirrhosis edema, abdominal swelling and ascites may appear, together with anemia and jaundice. Glycogen deposits also have been identified in the mucous glands of the gastrointestinal tract, but no specific changes referable to this have been reported.

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Adrenal Disorders.—Patients with adrenal cortical insufficiency have severe vomiting and dehydration, which may simulate high intestinal obstruction. Two such cases were reported by Weens and Golden. In one signs of hypertrophic pyloric stenosis were present, and in the second duodenal obstruction was simulated. In a discussion of radiologic changes seen with Addison's disease Jarvis, Jenkins, Sosman and Thorn reported adrenal calcifications in 24 out of 106 patients, but these were not considered definitely diagnostic. Gastrointestinal examinations were normal in 20 out of 24 cases. Two patients had peptic ulcers. In two others evidence of gastritis and superficial gastrointestinal ulcerations were observed, and the authors commented that such changes may be encountered during acute adrenal insufficiency. The peptic ulcers may have antedated the appearance of Addison's disease, and may have been activated during steroid therapy.

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VASCULAR DISORDERS

Hereditary Hemorrhagic Telangiectasis.—This is a familial disturbance, characterized by multiple localized areas of exposed thin-walled capillaries anywhere in the gastrointestinal tract as well as in other parenchymatous organs and the skin. Gastrointestinal bleeding may vary from traces of blood to massive loss. Bleeding occurs most often in the small intestine, and radiologic examination rarely discloses its source. However, cases have been described in which hematomas in the vicinity of the bleeding points caused intestinal obstruction. White and Wollenman reported a 6-year-old boy with intestinal obstruction due to a spontaneous subserosal hemorrhage from an angiomatous malformation associated with reduplication of the jejunum. Occasionally telangiectatic lesions are large enough to be mistaken for hemangiomas, but this is infrequent and few appear as actual tumors.

Hereditary telangiectasis should be differentiated from vascular malformations of the intestinal tract occurring as single lesions, even though they may be part of the same general disorder. A single telangiectasis of the colon with massive hemorrhage was reported by Rutter in a 31-year-old man who had to be operated upon 3 times before the bleeding site was found. This solitary telangiectasis of the colonic mucosa was considered similar to that of the congenital hereditary variety, but inasmuch as only a single lesion was identified there was some question as to whether this might be an isolated hamartoma.

Hemangiomas of the small intestine occur as diffusely infiltrating, circumscribed or polypoid lesions. These can produce intestinal obstruction or intussusception. The diagnosis is made at operation, but every now and then it is made radiologically because of the presence of phleboliths. Hollingsworth made a tentative diagnosis of hemangioma on the basis of numerous phleboliths in a child's pelvis even though these are unusual before adult life.

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Blood Dyscrasias.—Gastrointestinal symptoms are associated with *Henoch's purpura* quite often. The fundamental purpuric lesions occur as the result of an immunologic mechanism which produces an acute perivasculitis in the walls of the small vessels of the skin, the intestinal walls, the kidneys and the synovial membranes. This is the non-thrombocytopenic variety of purpura, with non-traumatic hemorrhages in the skin, subcutaneous tissues, joints and viscera. The cutaneous manifestations may appear after the gastrointestinal symptoms. Hemorrhage in the intestinal walls is followed by colicky pain, bleeding, constipation and vomiting. When abdominal symptoms appear before the cutaneous changes, acute disorders such as appendicitis, obstruction or a ruptured viscus may be simulated. The association of intussusception and Henoch's purpura gives rise to diagnostic difficulty. The two may coexist, and unless this is kept in mind the optimum time for reduction may be missed. Steinhardt and Jonas collected 24 cases of concomitant purpura and intussusception, and reported another in a 5-year-old girl. The cases of intussusception with Henoch's purpura are about equally divided between the ileocecal and ileoileal varieties, and occasional instances of colocolic intussusception also are known. Hematomas of the lesser bowel with consequent obstruction have been observed, and these may act as the leading part. Jones and Connell noted that gastric involvement may simulate inflammatory and ulcerative lesions, and that the small intestinal changes are those of disordered motor function with hypomotility, dilatation, segmentation and irregular mucosal markings. In a case reported by Fetter and Mills follow-up examinations over 3 months showed a normal small bowel pattern after the symptoms disappeared.

Gastrointestinal radiographic changes are uncommon with *hemophilia*. In a case reported by Lautkin, Korelitz and Berger a 9-cm. length of distal transverse colon presented thickened folds and constant lack of distensibility. Two months later this was normal, and it was postulated that intramural bleeding was responsible for the change, inasmuch as it had been associated with melena.

Polycythemia also is associated with gastrointestinal symptoms. Frequently these are due to thrombosis of mesenteric as well as intraintestinal vessels, and bleeding may be secondary to these thrombotic episodes. Involvement of the liver causes cirrhosis with its consequent chain of radiologic changes. Splenomegaly and hepatomegaly is fairly frequent with polycythemia vera.

The principle hemolytic anemias of infancy and childhood, including erythroblastic anemia, sickle cell anemia and spherocytic anemia, present little in the way of gastrointestinal manifestations. However, they are associated frequently with considerable splenomegaly. Calcification within the spleen secondary to sickle cell anemia was reported by Jacobson and Zuckerman in 4 out of 106 patients. These had deposits of calcium and iron which occurred as closely packed nodular densities. The spleen had been enlarged, but later diminished in size so that the deposits became irregular and confluent. The bony changes associated with erythroblastic anemia produce the most common radiologic manifestations of this disease, and skeletal changes also occur with sickle cell anemia, and to a lesser degree with familial hemolytic anemia.

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Mesenteric Vascular Occlusion.—Mesenteric vascular occlusion involves the arteries, veins or both. In most cases obstruction occurs in the superior mesenteric vessels, but occasionally the inferior mesenteric vessels are involved. Obstruction occurs either in an acute fulminating form or as a subacute process, the clinical syndrome varying with the intensity of the obstruction as well as with the impairment of the bowel wall. Blockage of the superior mesenteric vessels results in impaired blood supply to the lesser and large bowel as far as the distal transverse colon. Involvement of the inferior mesenteric artery impairs the bowel supplied by this trunk. The most fulminating type of this disturbance follows sudden and extensive obliteration of the superior mesenteric vessels. The greater incidence of such involvement is attributed to their larger

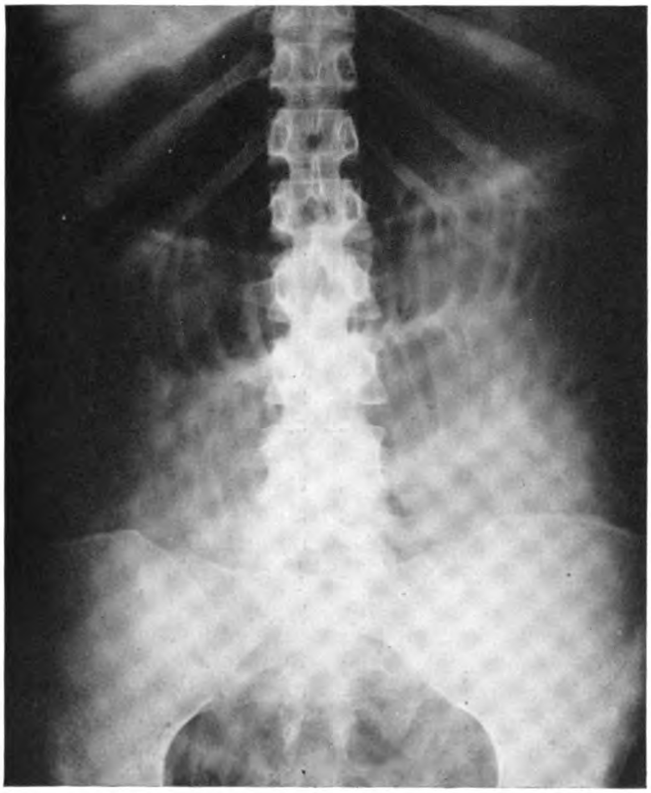

FIG. 206.—Mesenteric thrombosis in a 22-year-old woman. Marked ileus and gastric dilatation. No gas in the colon. At operation the jejunum was gangrenous, and peritonitis was present. Verified at necropsy

size and higher point of origin from the abdominal aorta, as well as to the fact that the superior mesenteric artery comes off practically parallel to the aorta. The inferior mesenteric artery is set at nearly a right angle to the aorta, and thus is less likely to intercept an embolus. Obstruction of the inferior mesenteric vessels is less likely to be serious because a collateral circulation can be established with the middle and inferior hemorrhoidal arteries below and the superior mesenteric artery above.

Obstruction to the vascular supply of the bowel occurs in any age group. Arterial obstruction may be associated with trauma, arteriosclerosis, thromboangiitis obliterans or the collagen disturbances. Venous thrombosis is seen with congestive heart failure, portal stasis and infection of organs which drain into the portal circulation. Pressure from intra-abdominal tumors also causes thrombotic changes occasionally. Certain blood disorders, particularly polycythemia, also are associated with this condition. Rarely primary portal phlebosclerosis and phlebothrombosis are the cause of extensive venous thrombosis. Embolic changes occur in patients with cardiac disease, particularly with vegetations on the heart wall or the aortic valves. Auricular fibrillation and chronic pulmonary disease also are causes for embolization to the mesenteric circulation.

Often it is not possible to make a definite diagnosis of arterial or venous obstruction. The episode may start acutely, with profound shock, or the onset may be gradual with vague symptoms for a day or two before the full effect of the occlusion is manifest. Hematemesis is observed in about 20 per cent of cases, and bloody diarrhea is fairly frequent. Diffuse colicky pain, distention and abdominal tenderness may lead to a diagnosis of intestinal obstruction. At operation the appearance of the bowel varies with the extent of vascular impairment. The lesser bowel may be pale and swollen, or if the obstruction has been present for some time, the loops become purple-black and distended with dark bloody fluid. The abdominal cavity may contain bloody fluid, and while perforation of the bowel is infrequent peritonitis is not uncommon. Ordinarily from 3 to 9 feet of jejunum or ileum are infarcted, and in most cases with the distal ileum involved, the terminal foot or so may be uninvolved because of individual variations in blood supply.

The radiologic picture of mesenteric vascular occlusion is not specific. The ileus which appears is variable. In some, distention is mild, with scattered loops of dilated bowel. Later a more diffuse and extensive ileus appears, often with fluid levels. Distention of the ascending and transverse colon appear fairly often. Barium enema often is required to eliminate the possibility of an obstruction in the splenic flexure or descending colon when bowel distention appears to stop abruptly. The use of barium by mouth was suggested by Frimann-Dahl, who found this useful for excluding intestinal obstruction. We have found the water-soluble unabsorbable contrast media useful for the same purpose without the disadvantages of barium.

The demonstration of a localized mesenteric thrombosis by the identification of a segmental dilatation of an ileal loop was reported by Cregg and Smith, who found that there was sufficient edema and hemorrhage in the bowel wall to render the loop clearly visible. In their patient, a 65-year-old man, a second episode of localized mesenteric thrombosis occurred 9 days after resection of an acutely infarcted segment of ileum. The preoperative roentgenogram at the time of the second thrombosis established the diagnosis by disclosing a distended ileal loop in the right upper quadrant. Similar radiographic changes occur with intramural mesenteric hemorrhage, intestinal strangulation, localized inflammatory disease and neoplasms with vascular or lymphatic obstruction.

Segmental infarction of the small intestine involves relatively short segments, which become partly devitalized rather than gangrenous. In some, healing is followed by stenosis. Wolf and Marshak reported 4 cases with small bowel infarction which went on to segmental stenosis and intestinal obstruction, the stenotic areas being from 3 to 6 inches long. Vascular changes in the wall of a strangulated loop of intestine caught in a hernia also may result in cicatricial tubular stenosis. The vascular impairment may be insufficient to cause gangrene, but enough to produce permanent injury of the intramural vessels. Later, infection produces cicatricial stenosis with obstruction. Radiographic examination shows the mucosa of these loops to be flattened, with a fairly abrupt and somewhat conical transition to normal dilated bowel proximally. Of the 4 cases reported by Wolf and Marshak one followed an incarcerated and strangulated hernia, and another a venous occlusion due to thromboangiitis obliterans. In the remaining two a similar radiologic appearance was noted, but the etiology was unknown aside from a presumption that the change was due to mesenteric occlusion. The removed specimen was a fibrotic, tubular stenotic loop which closely approximated the appearance suggested on the roentgenograms. Hawkins reported a 65-year-old man who survived an episode of gangrene without operation, after which a tubular stenosis resembling regional enteritis was found. This patient had had attacks of subacute intestinal obstruction due to the jejunal stenosis. At operation, this loop about 3 inches long, was situated about 6 inches from the duodenojejunal junction, and was grossly hard and thickened. Each of its ends terminated abruptly, giving place to normal bowel. Adhesions around it suggested the presence of previous inflammation. The episode was believed to have been due to embolization after a coronary thrombosis.

Infarction of the omentum is uncommon, and may present as a surgical emergency. Shea, Pomer and Spellman collected 37 cases and reported 6 more. The diagnosis is established at operation. No characteristic radiologic change is present, but ileus may be prominent.

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Aneurysms.—Aneurysms of the abdominal aorta and its main branches can produce gastrointestinal complaints. Most aneurysms are arteriosclerotic in origin, and occur mainly below the renal arteries as localized, saccular structures or diffuse dilatations extending into one or both iliac arteries. Symptoms vary from none at all to diffuse severe abdominal pain radiating to the back and groins. Much depends on the size of the aneurysm, its length and its pressure effects upon adjacent organs. With leakage of blood into the retroperitoneal area from dissection or perforation, severe pain and shock may occur rapidly. An aneurysm may affix itself to and rupture with exsanguinating hemorrhage into the stomach or the intestinal tract. Physical examination sometimes discloses a palpable mass. Inasmuch as aneurysms frequently are lined with thick laminated clot, pulsations often are absent. Thrombosis sufficient to impede the circulation to the lower extremities is associated with the Leriche syndrome.

Direct radiographic examination of the abdomen frequently reveals aneurysms because of mural calcification. These lime salt deposits are seen overlying and to the left of the lumbar vertebræ, and usually are better seen on lateral films. The posterior wall of many aneurysms remain relatively straight. Vertebral changes due to pulsations against the spine are occasionally seen in aneurysms of the lower thoracic and upper abdominal aorta, but are infrequent in the middle lumbar area.

Aneurysms impinging into the cardia produce a radiologic appearance simulating carcinoma or an extrinsic cyst. Those indenting the lesser curvature are associated with rigidity and diminished peristalsis. Rarely pyloric obstruction is found to be caused by fixation to an aneurysm. The duodenal loop sometimes is deformed by an adjacent aneurysm, with an appearance like that of a tumor of the head of the pancreas. Displacement of the left ureter and kidney also have been observed. Aneurysms occasionally extend into the renal arteries, or actually originate in these structures. Renal artery aneurysms may be identified by a calcific-lined density close to or within the hilum of the involved kidney, and distortions of the internal renal architecture and displacement of the adjacent ureter may be considerable. Diminished or absent renal function due to impaired blood flow sometimes is observed. Complete obstruction to the flow of blood causes infarction. Occasionally this occurs in a secondary branch, with infarction of half the kidney rather than the entire structure.

Aneurysms of the splenic artery frequently are seen on plain abdominal films in the left

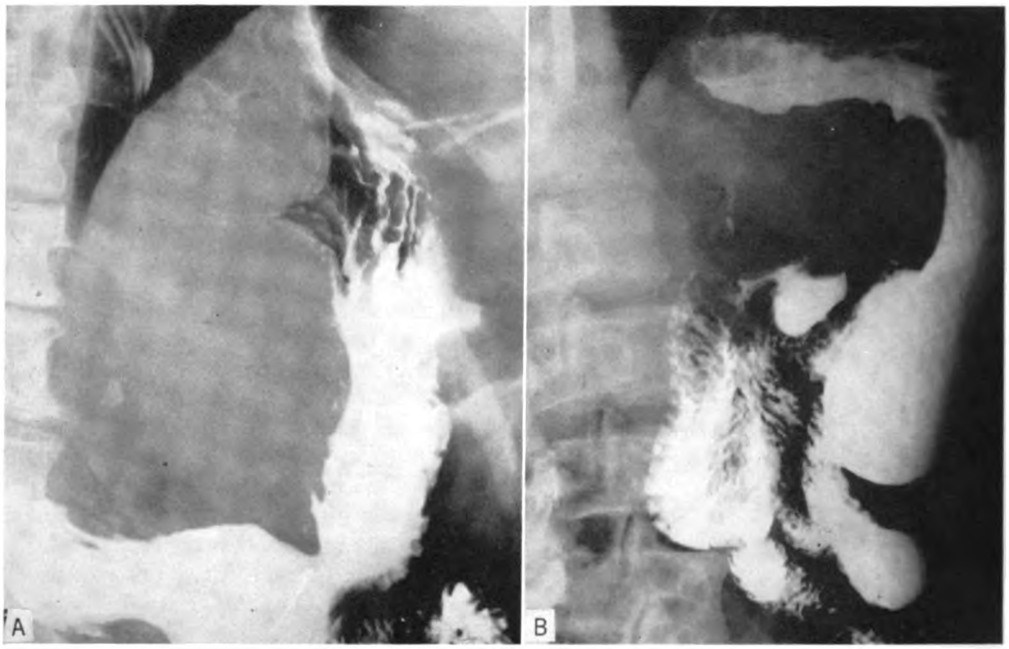

FIG. 207.—*A*, Aneurysm of abdominal aorta impressing into stomach gas bubble, simulating carcinoma. Verified at operation.

B, Aneurysm of abdominal aorta impressing against lesser curvature of stomach, verified.

hypochondrium. As a rule, these are asymptomatic, but occasional instances of rupture are known. Zeluff and Parsonnet reported such a happening in a 32-year-old woman. Sometimes these aneurysms are better demonstrated on body section roentgenograms, and percutaneous aortography also has been used in their identification.

Splenic artery aneurysms sometimes achieve sufficient size to produce a palpable pulsating mass in the left hypochondrium. These are identified by circular calcific rings rather than by the elongated cirsoid type of splenic artery aneurysm. The former variety has a greater potential for danger, and removal is considered advisable to safeguard against the possibility of rupture.

Aneurysms of the hepatic artery are uncommon, and their radiologic identification is rare. The diagnosis was made by Bruwer and Hallenbeck in a 60-year-old woman, and by Jarvis and Hodes in a 74-year-old man. In the first case the lesion was identified as a soft tissue mass intruding into the duodenal bulb. This was believed to be an ulcerating leiomyoma. At operation an aneurysm of the hepatic artery was found, which caused hemorrhage because of ulceration into the duodenal cap. In the second instance a multiloculated aneurysm was identified because of calcification within its walls. Jarvis and Hodes remarked that of the 88 cases reported the radiologic manifestations had been quite overlooked.

The clinical picture of hepatic artery aneurysm reflects their tendency to bleed or compress the bile ducts. Pain, hemorrhage, and intermittent obstructive jaundice are present. Ascites and a large pulsating liver also have been found. Rupture of the aneurysm is a fairly common terminal event. Malloy and Jason found that rupture of the aneurysm occurred in 67 out of 85 collected cases. Of these, 35 ruptured into the abdomen, 22 into the extrahepatic bile ducts, 7 into the duodenum, 6 into the gallbladder, 1 into the portal vein and 1 into the stomach.

Aneurysms of the hepatic artery follow infection, arteriosclerosis or trauma. Syphilis is not a prominent factor. Most are extra-

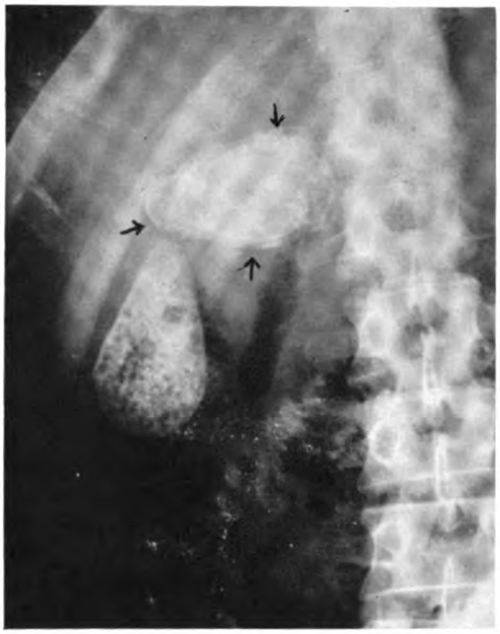

FIG. 208.—Aneurysm of hepatic artery (arrows), verified at operation. Oral cholecystography shows a functioning gallbladder full of stones.

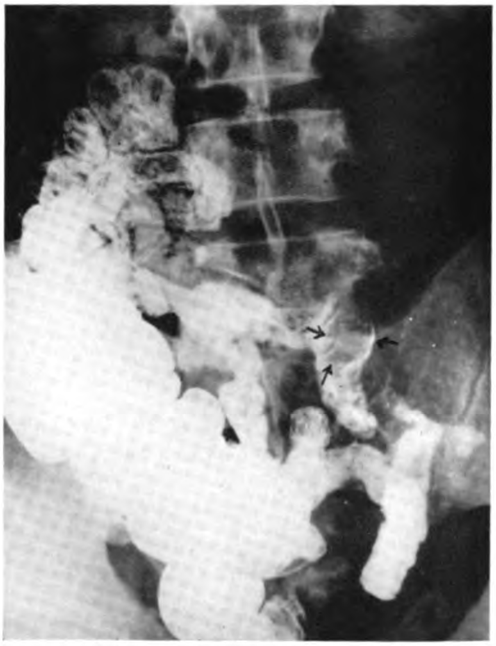

FIG. 209.—Aneurysmal tumor of jejunum, verified at operation, in a 42-year-old man with weakness and melena. Small intestinal study reveals a small circular defect (arrows) in the jejunum overlying the upper sacrum.

hepatic, but intrahepatic dilatations also occur. These aneurysms are most often single saccular dilatations. The main trunk of the hepatic artery is affected mainly, but the right main trunk and to a lesser extent the left main trunk also may become aneurysmal. The larger sacs in part owe their size to leakage and consequent hematomas, which then impinge on adjacent structures.

One verified case of hepatic artery aneurysm was observed by us in a 35-year-old man. The plain films of the abdomen disclosed a calcareous, egg-shaped mass in the right hypochondrium. This measured about 7 cm. in length and 4 cm. in width. At first this was believed to represent an echinococcus cyst. Oral cholecystography disclosed a gallbladder packed full of stones, quite separate from this mass. At operation a large calcified hepatic artery aneurysm was identified, and left undisturbed inasmuch as no symptoms were referable to it.

Mycotic aneurysms follow implantation of septic emboli into the arterial wall, producing small lesions which are not identifiable radiologically. They also appear after the entrance of an infected embolus, with spreading of infection to the adjacent walls after thrombosis. These lesions are asymptomatic, but occasionally a vessel becomes fixed against the stomach or intestine, and perforates with severe hemorrhage. Zarowitz and Grayzel reported a patient with tuberculous arteritis of the abdominal aorta which ruptured into the third portion of the duodenum.

Small congenital aneurysms also appear in the gastrointestinal tract. While often completely asymptomatic, they may cause bleeding. In a verified case seen by us, an aneurysm about 1 cm. in diameter was identified radiologically in the distal jejunum as a "mass lesion." Rathmell, Horwell and Greeley reported a 29-year-old woman in whom a fatal intestinal hemorrhage was due to a congenital jejunal aneurysm. In this patient a minute search of the bowel mucosa was required before a small mucosal polypoid lesion was found projecting into the lumen of the jejunum. This was about 5 mm. in diameter, and presented as a smooth gray glistening lesion. Histologic examination disclosed a somewhat collapsed, thick walled, vascular lesion, one edge of which had perforated.

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Chapter 4

Traumatic Disorders, Foreign Bodies and Effects of Drugs

TRAUMATIC DIAPHRAGMATIC HERNIAS

THESE are more frequent on the left side, and follow either penetrating or nonpenetrating wounds of the chest and abdomen. They were particularly frequent during World War II, and also appear after accidents. Acute symptoms are due to hemorrhage, mediastinal shift or injury of displaced bowel or viscera. However, symptoms often are delayed, and sometimes the condition does not become apparent for a long time after the injury. Traumatic diaphragmatic hernias are not true hernias because no peritoneal sac is present. The viscera passes upward into the left thoracic cavity, and structures as diverse as the stomach, bowel, omentum, spleen, pancreas, kidney and liver have been found in the left hemithorax. No direct correlation has been established between any anatomic weakness of the diaphragm and the location of the tear. It is believed that increased intra-abdominal pressure is an important contributing cause of herniation through small defects which may have been caused by previous injury, or be of congenital origin. Traumatic right diaphragmatic hernias are relatively uncommon because of the protecting action of the liver. Nevertheless, in some cases a tear in the right diaphragm may be large enough to permit entry of the liver into the right chest

The clinical picture of traumatic diaphragmatic hernia often is bizarre. In some cases no symptoms are apparent, while in others the complaints suggest cardiac or respiratory disorders. Symptoms of intestinal obstruction or strangulation are sometimes sudden in onset and require surgical relief. Strangulation may be obscured by a bloody pleural effusion.

Radiographic examination of the chest is diagnostic when gas-containing stomach and intestine are in the thoracic cavity. The mediastinal contents and heart may be displaced to the right. The diagnosis usually is established clearly by giving the patient a barium meal, and following it for 24 to 48 hours to determine the position of the entire gastrointestinal tract. There may be a high, arcuate shadow simulating an elevated left diaphragm which is produced by dilatation of the stomach displaced into the left chest. This may be apparent in the lateral as well as in the frontal projections, and sometimes is mistaken for eventration. The possibilities for strangulation of gut in the thorax is increased if the defect in the diaphragm is relatively small, permitting rotation and pinching off of the bowel passing through it. Such an occurrence may require barium enema examination to disclose the location of an obstruction in the vicinity of the left diaphragm. If stomach or lesser bowel become strangulated the signs will be those of high obstruction or absence of gas in the lesser bowel, depending upon the involved site.

The increasing use of thoracic surgical approaches to the abdomen is associated with entrance of the stomach into the chest through wounds which fail to heal properly. The uppermost wall of the stomach then simulates an elevated diaphragm. When gastric dilatation becomes extensive abdominal and thoracic pain, dyspnea, tachycardia, fever and sometimes shock follow. Strangulation requires surgical relief. Mention of herniation following operative incision of the left diaphragm was made by Carter, Giuseffi and Felson, and another case was reported recently by Loitman, Hoover, Miscall and Evans. In a recent case seen

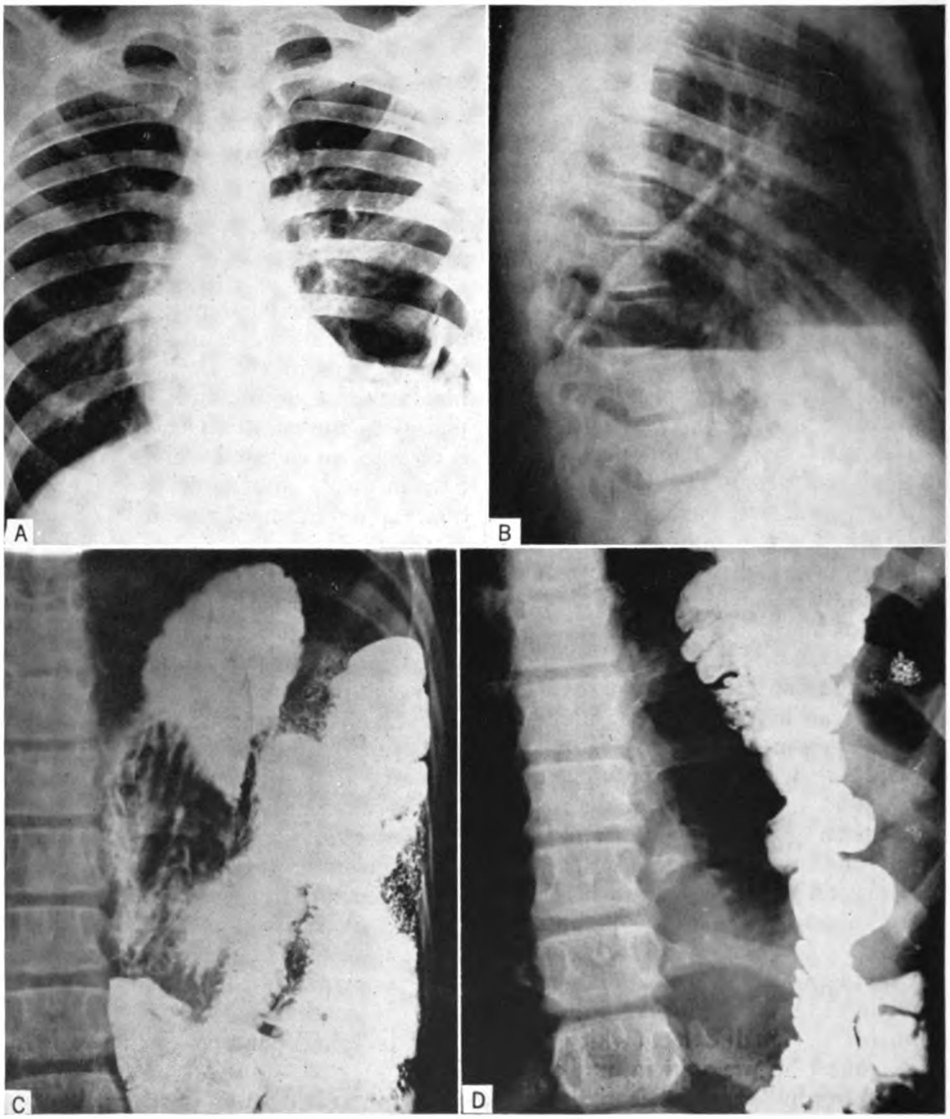

FIG. 210.—*A*, Post-traumatic diaphragmatic hernia in a 13-year-old boy. Onset of symptoms 3 years after automobile accident with dyspnea and epigastric pain after exercise. Chest film shows gas-filled viscus in left chest.

B, Same patient, lateral view.

C, Same patient, gastrointestinal examination shows stomach and lesser bowel in left chest.

D, Same patient, barium enema shows colon in left chest.

by us gastric herniation into the left thoracic cavity followed operation for achalasia of the esophagus. On postoperative films the left diaphragm appeared high. The patient complained of difficulty in breathing and chest pain. The diagnosis became evident after the patient was given some water-soluble contrast medium to drink. This revealed that the stomach wall was the shadow previously thought to be the elevated diaphragm.

INCISIONAL HERNIAS

Entrance of bowel or omentum into hernias protruding through ununited or poorly

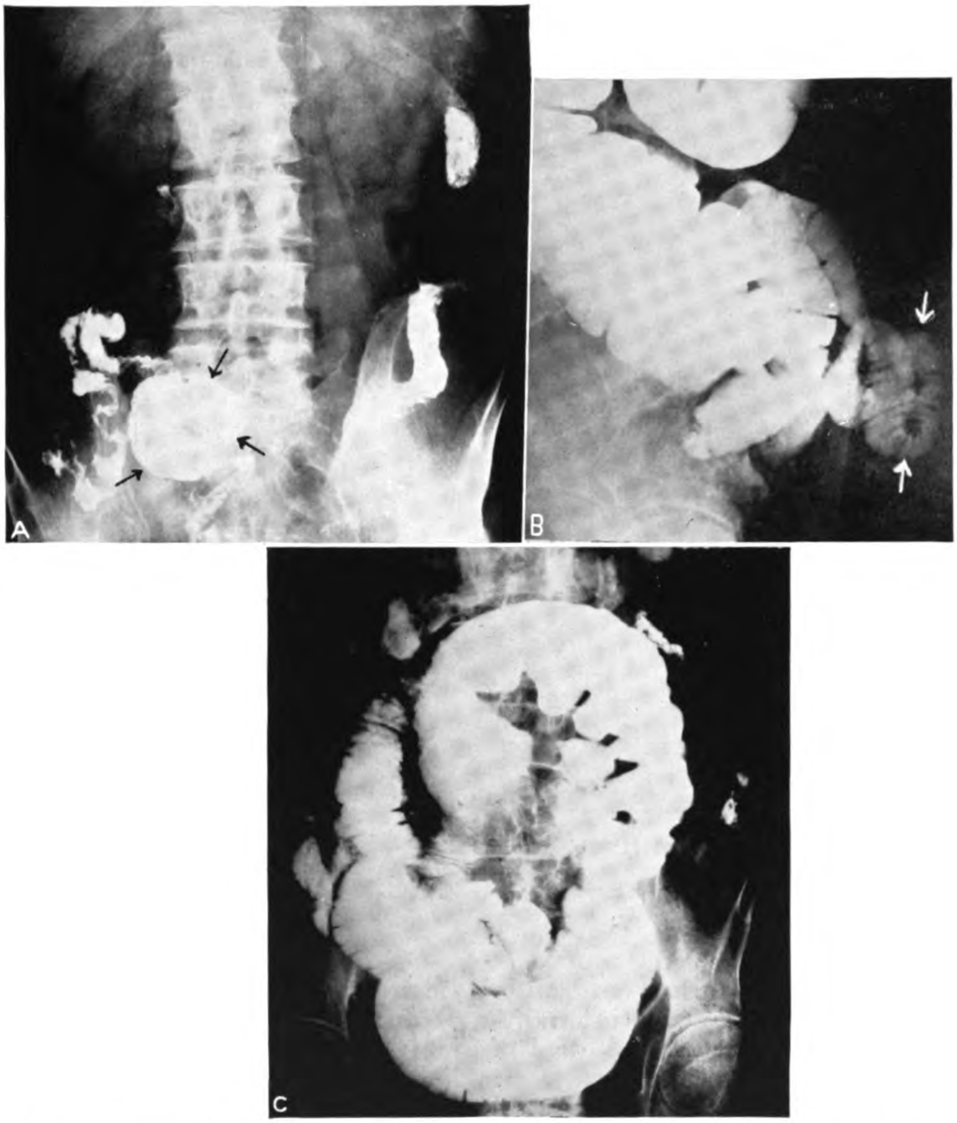

FIG. 211.—*A*, Incisional hernia after prostatectomy in a 70-year-old man. Post-evacuation study shows coiled loop of intestine fixed in position (arrows) in lower abdomen.

B, Conedown lateral of barium enema, same patient. A loop of small intestine protrudes beyond anterior abdominal wall (arrows).

C, Same patient, 5-hour film, gastrointestinal examination reveals obstruction in midileum.

united surgical incisions were more common several decades ago, particularly in patients who had had vertical abdominal incisions. These sometimes were enormous and most uncomfortable. With large openings, these sacs contained lesser and large bowel, and sometimes the kidney and spleen also entered the hernia. So long as no bowel obstruction was produced, the symptoms were mainly discomfort and easy fatiguability. However, partial intestinal obstruction appeared because of adhesions and rotation of bowel loops. In some, obstruction became complete and strangulation complicated the situation.

Incisional hernias are quite obvious on

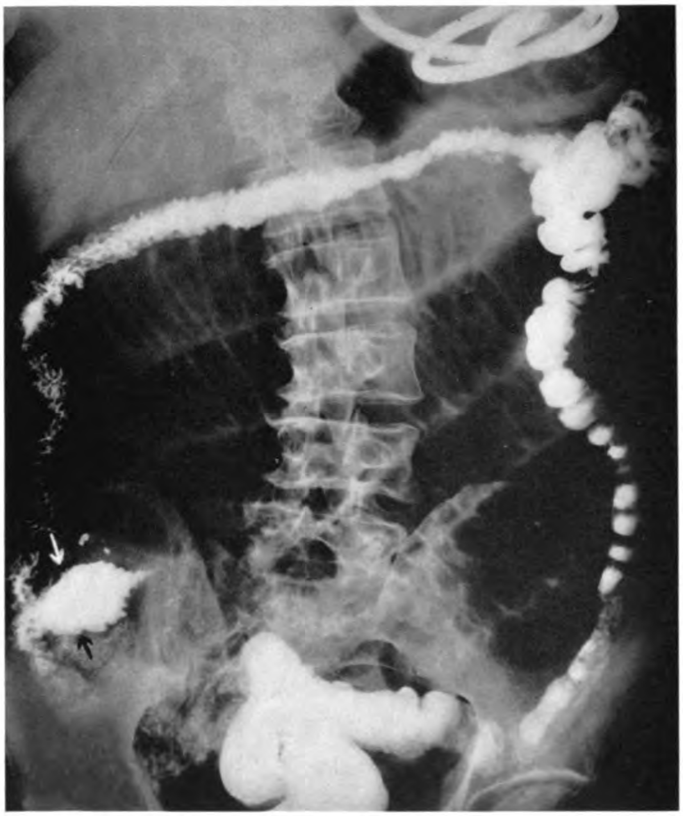

FIG. 212.—Richter's hernia in a 57-year-old man with fecal vomiting and abdominal cramps. Post-evacuation film shows colon to be normal. A knuckled loop of ileum (arrows) is caught in a mesenteric fold in the right lower quadrant, with dilatation of the proximal bowel. Verified at operation undertaken for intestinal obstruction.

physical examination. However, gastrointestinal examinations are useful in identifying the contents of the sac. A word of warning is indicated when barium enema examination is contemplated. If the bowel does not empty adequately, considerable discomfort may follow retention of barium in the bowel within the hernia.

A Richter's hernia is rarely recognized radiologically, but must always be considered in the diagnosis of obscure obstructive lesions of the small intestine. This type of hernia occurs when the antimesenteric surface of the bowel protrudes into an intra-abdominal orifice, or is caught in a tunnel of adhesions. Similar protrusions occur in ventral and umbilical hernias. The usual site is at the femoral canal. Knuckling of the incarcerated loop produces a partial enterocele with congestion and edema of a portion of its wall. Adhesive bands connecting the hernial sac with the incarcerated loop may prevent reduction of the involved bowel. As a result of this, a diverticulum-like deformity is produced which causes congestion, edema and ultimately intestinal obstruction. Nausea and vomiting, together with a palpable tender mass or local tenderness over a hernial orifice or incisional site provides a clue to this diagnosis. Radiological investigations usually disclose nothing characteristic. Gillespie, Glas, Mertz and Musselman reported that symptoms of intestinal obstruction were present in somewhat less than half their patients. In the others, the abdominal distention together with nausea and vomiting sometimes suggested the diagnosis.

This is a deceptive condition, and while not frequent, is of great importance because operation in time may be life-saving. A complication is enterocutaneous fistula, with subsidence of nausea, vomiting and distention as the fistulous tract serves to relieve obstruction.

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ABDOMINAL TRAUMA

Direct trauma may be either penetrating or non-penetrating, or have elements of both types of injury. The penetrating form is an obvious indication for proper surgical management as soon as possible. As a rule, these patients do not come to radiologic investigation. In some, scout abdominal films may be required to disclose pneumoperitoneum, and radiologic evidence of bleeding into the abdomen and retroperitoneal areas may be helpful in evaluating the extent of the injury.

Non-penetrating trauma with the abdominal walls intact presents a diagnostic challenge both to the clinician and the radiologist. The possibility of injury to parenchymatous organs must be kept in mind constantly. While the injury may be limited to a single organ, the presence of multiple injuries must not be overlooked. Too great reliance should not be placed on so-called "negative" radiologic examinations, and clinical acumen often is of far greater importance than reliance on radiographic methods.

In a review of 47 patients with non-penetrating abdominal trauma Pontius, Kilbourne and Paul found injury to single organs in 84 per cent of their cases. Of these, the spleen was involved in 36 per cent, the liver in 12 per cent, and the gastrointestinal tract in 36 per cent. Injuries of the jejunum and ileum occurred in 10 of 15 cases, with rupture of the colon in 3, the duodenum in 1 and the stomach in 1. Seven patients had multiple injuries of various segments of the small and large intestine, and rupture of various large vessels. There were 5 unproved cases in this group. The force of the trauma may not be parallel to the extent of the injuries, and minor trauma often causes a major disturbance. In some patients abdominal trauma is overlooked because of more obvious injuries to the head, neck, chest or extremities. The history often is unreliable, particularly in children, and evidence of injury to the spleen, liver and kidneys must be sought by careful examination and well-planned radiographic study.

Symptoms associated with abdominal trauma often are dramatic, with shock and severe pain dependent upon the location and severity of the injuries. Abdominal muscular rigidity and tenderness, blood in the stomach or the bowel, pain in the shoulder or testes all point towards specific injuries. Direct abdominal trauma often produces ileus. Similar intestinal distention also is produced by injuries to the thorax or extremities.

From the radiologic viewpoint, direct examination of the abdomen after trauma is important in the identification of pneumoperitoneum, which may indicate the presence of a ruptured viscus, particularly the stomach or bowel. However, absence of pneumoperitoneum does not exclude small intestinal perforation. Traumatic rupture of the duodenum need not be accompanied immediately by severe symptoms, and the apparent comfort of the patient may last as long as 12 hours. Perforation of the duodenum is accompanied by retroperitoneal emphysema, with extension of the air to the vicinity of the right kidney. Retroperitoneal emphysema extending along the psoas muscles sometimes involves the left renal area as well. A retroperitoneal hematoma can obliterate the psoas and kidney shadows. A similar picture sometimes is produced by rupture of the retroperitoneal aspect of the ascending colon. The mechanism for duodenal or colon rupture is a crushing force against the vertebræ or a tangential tearing of fixed retroperitoneal attachments. The forceful propulsion of gastric contents through an open pylorus into the duodenum also may be a factor in the production of a duodenal rent. Retroperitoneal emphysema is manifested by a bubble-like collection of gas in the soft tissues, which presents an appearance quite different from that associated with free gas in the peritoneal cavity.

Rupture of the small intestine can occur anywhere along the jejunum or ileum. As a rule, these perforations are small. In the 20 cases reported by Geoghegan and Brush, the holes varied from 4 mm. to 2 cm. in 10 cases in which the sizes were reported exactly, and in 6 others the tears were considered small. Because of this, peritonitis often appears slowly, and the radiologic changes are not striking if the patients are examined soon after the injury. If peritonitis becomes extensive a pronounced adynamic ileus appears. Otherwise, the extent of the ileus may be only moderate, and often cannot be distinguished from that due to a trauma without bowel rupture. The passage of air into the peritoneal cavity is manifested by pneumoperitoneum. The use of contrast materials for the identification of lacerations was suggested by Schlosser and Harkins, who advised the injection of small quantities of lipiodol into nasogastric tubes for the purpose of visualizing duodenal lacerations otherwise undetectable. It would appear that the use of water-soluble contrast substances might find some merit here. The important thing to remember in patients who have had abdominal trauma is not to place too great reliance on the radiologic changes.

Perforation of the colon may be marked by the passage of feces into the peritoneal cavity. Gottlieb, Sharlin and Peck described 2 cases in which fecal shadows outside of the colon did not conform with the expected outline of the large intestine. They regarded this as a useful sign even in the absence of free gas in the peritoneal cavity.

Intramural hematomas of the bowel wall sometimes occur spontaneously, but more often follow trauma. They may be accompanied by symptoms of partial, or even complete intestinal obstruction. Such hematomas in the duodenum after trauma have been manifested radiologically as sharply demarcated tumefactions narrowing the lumen of the bowel, associated with a coiled-spring mucosal pattern. Cases such as these have been described by Felson and Levin, and by Melamed and Pantone. Intrinsic duodenal obstruction due to a submucous hematoma because of erosion of a small blood vessel incident to acute pancreatitis was described by Oppenheimer. The presenting sign was huge gastric dilatation. The pancreatitis was believed to have been initiated by rupture of dilated ducts behind a pancreatic carcinoma, or by bleeding from a vessel in the duodenal wall eroded by infiltrating carcinoma.

A traumatic intramural hematoma of the lesser bowel also can cause intestinal obstruction. Moody observed that in the 9 cases in the American Literature the hemorrhage had been caused by trauma which pinched the fixed retroperitoneal portion of the duodenum against the spine, disrupting the mesenteric attachment. The intramural hematoma which formed separated the serosa from the muscularis, producing a bulging mass which caused some obstruction. Liverud reported a 3-year-old boy who had been kicked in the abdomen, following which a mass was felt in the abdomen. This caused partial obstruction to the passage of barium, and proved to be a subserous hematoma in the jejunum. Obstructing hematomas of the large intestine were reported by Jones and Settle, and by Kratzer and Dixon in patients who had a history of abdominal injury followed several months later by a constricting lesion of the colon.

Hematomas of the rectus sheath can produce soft tissue shadows over the abdomen simulating an intra-abdominal tumor. In Mizbah's case, the mass was believed to be a mesenteric cyst. The roentgenograms showed dilated jejunal loops which appeared to surround the tumor partly. At operation a hematoma was found between the layers of the rectus sheath. This was attributed to rupture of blood vessels during a severe bout of coughing.

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RUPTURE OF THE SPLEEN

The clinical picture of traumatic rupture of the spleen depends on the type of hemorrhage produced. With a history of injury, pain in the left upper quadrant and a palpable spleen is most important in this diagnosis. When bleeding occurs immediately, the clinical appearance of shock and upper abdominal pain radiating to the left shoulder may be so conclusive that radiologic investigation is not required. However, a much less dramatic picture follows delayed hemorrhage or slow bleeding into the left hypochondrium due to slight capsular tears. An intrasplenic hematoma may go unnoticed for a considerable time. Sometimes a severe trauma is not associated with any demonstrable effect, while a relatively mild injury causes a significant splenic laceration. Hence, due cognizance must be given the possibility of splenic rupture even with slight injuries, particularly in children.

Radiologic investigation of the abdomen cannot be relied upon for the diagnosis of splenic rupture. There are certain signs which may be contributory, such as obscurance of the splenic contour by overlying blood, irritation of the left diaphragm with elevation and restriction of its motion, and irregularity of the greater curvature of the stomach due to extrinsic pressure from an adjacent sanguineous collection. Hemorrhage at the top of the spleen may displace its tip downwards. Sometimes it is helpful to fill the stomach with air or contrast material to help determine the presence of irregular filling defects along its greater curvature. Hemorrhage passing into the retroperitoneal tissues may produce obliteration or haziness of the left psoas and kidney shadows. Blood gravitating along the gastrosplenic ligament also can produce a serration of the greater curvature. Adjacent viscera such as the splenic flexure and kidney may be displaced. As an additional effect of trauma, gastric dilatation and ileus is not uncommon. The presence of blood in the peritoneal cavity may contribute to further intestinal distention.

With slow leakage of blood from a ruptured splenic capsule, the signs are delayed, and if bleeding stops spontaneously, they may disappear. Intrasplenic hematomas produce a gradual increase in the size of the spleen, with no other visible disturbances. These patients sometimes develop splenic cysts, with later calcification of the cyst walls.

The spleen frequently is not visible under normal circumstances. Such an observation, even in the presence of known trauma, is not a reliable diagnostic sign. However, if a sharply outlined spleen is identified, and the adjacent soft tissues are normal, the diagnosis of a ruptured spleen with significant bleeding becomes unlikely.

The diagnosis of splenic rupture by pneumoperitoneum has been suggested. This technic may reveal irregularities of the splenic outline suggestive of subcapsular hematomas, or may reveal free intra-abdominal fluid. If pneumoperitoneum is decided upon, it is advisable to precede the examination with erect and recumbent studies to make sure that a spontaneous pneumoperitoneum is not overshadowed by the artificially induced pneumoperitoneum.

Rupture of the spleen also occurs on a non-traumatic basis, particularly in patients with splenic disorders. Spontaneous intrasplenic hemorrhage occurs with infectious mononucleosis, malaria and various blood dyscrasias, and may be followed by the formation of hemorrhagic cysts which reach considerable size and displace the adjacent organs. Gipson, Chancey and Czernobilsky noted that splenic rupture might be precipitated by even slight trauma in patients with infectious mononucleosis. In a review of the literature they noted that 44 cases of splenic rupture complicating this disease had been described. Their case was the first large hemorrhagic cyst of the spleen complicating infectious mononucleosis to be reported. Enlarged spleens associated with chronic malaria, thrombosis of the splenic vein, typhoid fever also are prone to spontaneous rupture or to rupture after slight trauma. Moore observed non-traumatic rupture of the spleen during pregnancy as an unusual occurrence. He found 28 recorded cases and added 4 more.

Torsion of the spleen with pneumoperitoneum was described by Falor and Angus in 2 patients who gave short histories of severe left upper abdominal pain. The spleens were removed because of infarction. It was believed that a congenital factor was present, the pedicles being elongated and the configuration of the abdominal cavity such that rotation of the spleen about the pedicle could occur.

An interesting rare complication of splenic injury is dissemination of splenic tissue within the peritoneal cavity, which may give rise to intestinal obstruction.

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RUPTURE OF THE LIVER, BILIARY TRACT AND PANCREAS

The mortality of traumatic rupture of the liver is high because of massive hemorrhage. As a rule these patients do not come to radiologic examination. In some cases, trauma produces intrahepatic hemorrhage with passage of blood into the biliary system. In such cases a puzzling picture of hemorrhage from the biliary tract manifested by gastrointestinal bleeding or biliary colic follows. Trauma to the liver also produces cysts, which develop slowly and sometimes reach sufficient size practically to replace the right lobe.

The radiologic manifestations of traumatic rupture of the liver include obliteration of the liver shadow, signs of free blood within the peritoneal cavity and diffuse ileus. Hematomas of sufficient bulk displace the adjacent viscera and elevate the right diaphragm, splinting its motion. Should the diaphragm be torn, the liver may herniate into the right thoracic cavity. Pulmonary and pleural changes follow bleeding and associated lung damage. Penetrating abdominal wounds causing direct communication between the biliary tract and the thorax give rise to pleuro-biliary or broncho-biliary fistulas, with extravasation of bile into the pleural cavity forming a bilithorax. Communication with the bronchial passages is followed by expectoration of bile, and this usually is accompanied with rapid necrosis of the lung. Bronchography is useful in identifying bronchobiliary fistulas. These wounds are uncommon in civilian life, but are quite frequent in military circles.

Direct abdominal trauma results in temporary non-visualization of the biliary tract with oral or intravenous cholecystography. This may follow injury to any part of the trunk, and occurs when hepatic function tests are normal. The duration of the period of non-visualization depends on the extent of the injury.

Traumatic rupture of the bile ducts may be associated with acute hemorrhage pancreatitis because of passage of bile into the pancreas through a laceration. The entrance of bile into the peritoneal cavity produces biliary peritonitis, sometimes with accumulation of large amounts of bile-stained fluid. Contusions and lacerations of the pancreas releases pancreatic juice with consequent peritoneal irritation, and retroperitoneal inflammations and fluid collections may also appear. Bizarre changes such as practically complete dissection of the gallbladder from its bed or transection of the pancreas depend on the traumatic forces and their direction of impact. The clinical picture associated with these injuries often are so urgent that radiologic examinations are not done.

With delayed reactions to abdominal trauma, the diffuse pancreatitis may become chronic, with later development of calcifications and cysts. Pancreatic fistulas also follow injuries to the upper abdomen, with later formation of subphrenic and retroperitoneal abscesses.

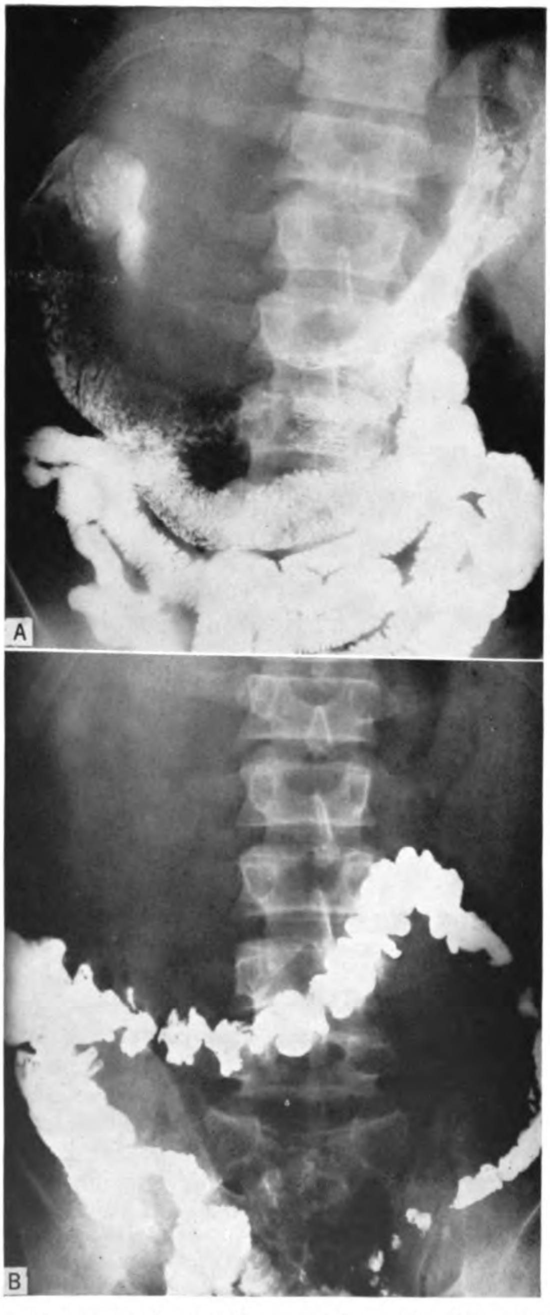

Fig. 213.—*A*, Post-traumatic pancreatic cyst diagnosed 5 months after trauma. Note displacement of stomach and duodenum.

B, Same patient. The transverse colon is depressed as well.

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ADVERSE EFFECTS OF CERTAIN DIAGNOSTIC AND THERAPEUTIC PROCEDURES

With the advent of the introduction of radiopaque contrast media into the spleen or liver for splenoportography or transhepatic cholangiography, a certain number of adverse effects may be anticipated even under the best of circumstances. These may be bleeding into the peritoneal cavity from a laceration of the splenic capsule, or drainage of bile from a tear of the liver capsule. Such effects often are rather slow in their appearance, but nevertheless can cause serious consequences requiring surgical intervention. No specific radiographic changes are identified in these patients other than ileus secondary to the presence of blood or bile in the peritoneal cavity. If bleeding is extensive the scout abdominal films may reflect the accumulation of blood in the perisplenic or perihepatic areas.

The introduction of a trocar into the abdomen for removing ascitic fluid is a frequent procedure. However, care should be exercised to avoid introducing the trocar into an area which has been entered surgically. In such cases there may be adhesion of intestines against the abdominal scar. In 1 case seen by us the paracentesis was followed by development of a local abscess, which in turn caused intestinal obstruction.

Diagnostic pneumoperitoneum occasionally may be complicated by spontaneous pneumothorax, as reported by Repa and Jacobson. In their patient, thread-like shadows were noted on the superior surface of the involved diaphragm prior to actual rupture. It was believed that these shadows represented a separation of the muscle fibers of the diaphragm by the dissecting air. Therapeutic pneumoperitoneum sometimes is accompanied by diaphragmatic pneumoceles which appear as annular shadows, and which may be completely asymptomatic. However, these pneumoceles may rupture and produce tension pneumothorax.

Pneumoperitoneum sometimes occurs spontaneously, although it is well recognized that the most common cause is a perforated ulcer or a ruptured viscus. However, it may appear with no apparent cause and without symptoms. One possible cause for spontaneous pneumoperitoneum in women is vigorous knee-chest exercise, permitting the entrance of air into the abdomen by way of the fallopian tubes. Lozman and Newman reported that they had observed this in a patient performing postpartum exercises, an opinion concurred in by Conn and La Fon. Rarely, a newborn infant may present pneumoperitoneum with no underlying defect or injury. Porter reported a newborn infant delivered by cesarean section who had free intra-abdominal air, together with a normal distribution of intestinal gas and no evidence of obstruction. At operation free air was found in the peritoneal cavity, but no explanation for this could be found. Two similar cases had been reported in the literature.

Pneumoperitoneum may follow perforation of the stomach or esophagus during esophagogastroscopy. An interstitial gastric emphysema due to entrance of air into the wall of the stomach in a few cases has been followed by passage of air into the peritoneal cavity. As a rule, this is relatively infrequent, and is asymptomatic. Myrhe and Wilson noted that out of 119 cases in which roentgenograms of the abdomen had been made 24 hours after gastroscopy, there were 2 with interstitial air in the stomach wall.

This may be due to extravasation of gas through a weak point in the mucosa leading into the perivascular sheaths and thence into the serosa. Sometimes gas infiltrates along the peritoneal reflection of the stomach, entering the gastrohepatic ligament and omentum, and then ruptures into the peritoneal cavity. The gas also may enter the mediastinum and the retroperitoneal tissues, and pass as high as the neck or as low as the scrotum. The presence of gastric or esophageal neoplasm supposedly makes this complication more likely.

Perforation of the small intestine by a Miller-Abbott tube was reported by Berger and Achs, occurring in a patient under treatment for intestinal obstruction. Symptoms had been controlled for about a week, after which time drainage diminished and the patient's condition worsened. Operation disclosed that the tip of the tube distal to the balloon had protruded through the bowel wall just proximal to the point of obstruction. Occasionally a nasogastric tube passes beyond control. It takes from 1 to about 30 days for the tube to pass. Arrest of the tube is considered presumptive evidence of block. Intussusception may be due to the balloon acting as an invaginating mass. McKenzie, Moore and Miller described perforation of necrotic ileum by the balloon of a Miller-Abbott tube, with production of a pelvic abscess. Multiple perforations of the small bowel due to Cantor tubes also have been noted.

Rupture of the colon during or after expulsion of a barium enema is observed occasionally in patients who have obstructing neoplasms. It is probable that in some a preexisting perforation may have been aggravated by the force of the enema. The entrance of barium into the peritoneal cavity is associated with the production of adhesions in those who survive. Retroperitoneal emphysema occasionally occurs because of perforation of the bowel. Culver and Concannon described a case of perforation of a sigmoid carcinoma into its mesentery, with pneumoretroperitoneum. The air was distributed over the upper pole of the left kidney and adrenal, and passed between the diaphragm and the parietal peritoneum bilaterally. Retroperitoneal emphysema from perforation of the colon or lesser bowel is uncommon, but must be considered in differential diagnosis whenever this condition is identified. Another point in differential diagnosis is the occurrence of retroperitoneal emphysema which follows caudal anesthesia. Recently we observed a patient in whom a well-defined streaking of gas was seen in the psoas areas, extending up toward the kidney. In the absence of an adequate history one was entitled to inquire whether the retroperitoneal emphysema might not have been caused by perforation of a gas-containing viscus. On discussing the films with the physician in charge, it was soon apparent that the point of entrance of the air was the needle introduced 2 days before when a caudal block was performed.

Rupture of the colon in infants during barium enema may follow traumatic introduction of a rectal tube, particularly when rectal stenosis is present. In children without congenital malformations, rupture of the colon may occur because of distension of a rectal retention balloon. Under such circumstances, a longitudinal tear may appear, permitting entrance of stool and barium into the abdominal cavity. Hartman and Hills reported 2 cases, 1 in a 4-day-old infant and another in an 8-month-old child. Traumatic rupture of the rectum without injection of contrast materials also occurs, and a peritonitis may develop which can be diagnosed only at operation. If this is suspected, studies for the presence of pneumoperitoneum are helpful in clarifying the problem. Additional questioning may disclose an awkward attempt at taking a rectal temperature or some other reason for a minute perforation of the bowel.

The radiographic demonstration of intraperitoneal barium sometimes escapes notice during fluoroscopy. However, on the films the presence of barium along the lateral walls of the abdominal cavity outside of the bowel presents a characteristic picture.

Barium can enter the venous tree, with massive and sometimes fatal embolism. Such a case was described by Roman, Wagner and Steinbach in a 73-year-old man in whom a Bardex tube had been used. It was postulated that with straining a fissure opened, permitting barium to enter the

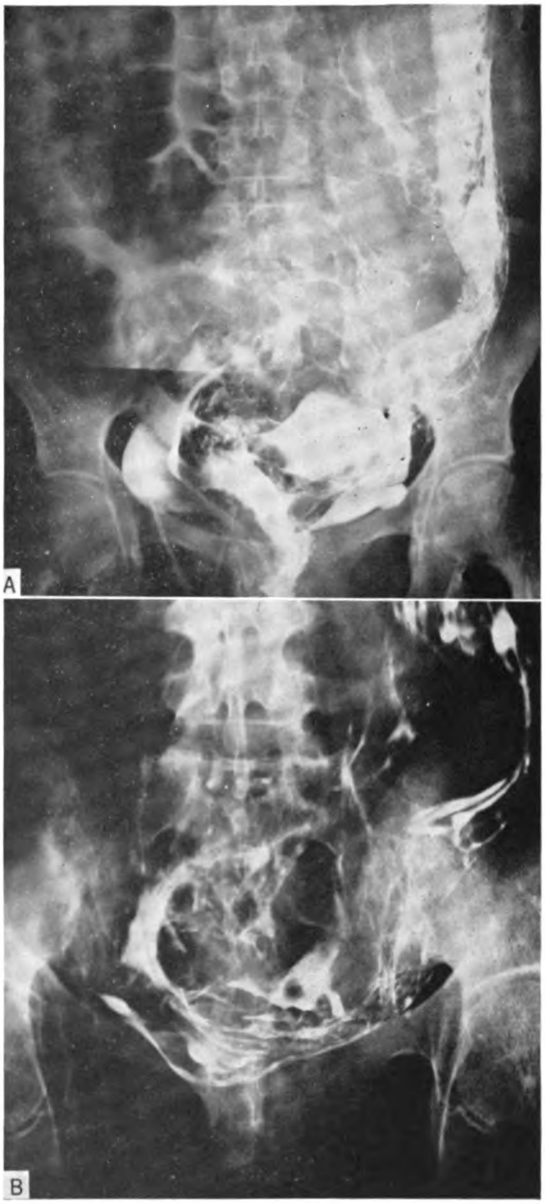

FIG. 214.—*A*, Rupture of sigmoid just above peritoneal reflection occurring during barium enema. Note the barium along the left lateral peritoneal wall.

B.—Same patient, 5 minutes later. The barium has spread throughout the peritoneal cavity.

venous system. Entrance of barium into the portal vein terminating in an intrahepatic abscess was described by Isaacs, Epstein and Nissen in a young woman with colitis.

Rupture of the colon during hydrostatic reduction of intussusception is uncommon, but must be kept in mind when there is a possibility of an irreducible lesion. This was discussed by Leichtling and Demetriades, who presented a 3-month-old infant in whom perforation and entrance of barium into the peritoneal cavity was noted during fluoroscopy. This patient was operated upon and the specimen disclosed entrapped ileum which was infarcted and ulcerated.

Occasionally a duodenal ulcer perforates during examination, with passage of barium into the abdomen. One never knows whether or not the perforation existed before the examination had been started. In some patients the entrance of barium is slow, presenting only as a serpentine shadow. In others a considerable quantity enters rapidly. The question as to whether barium alone is capable of producing an intraperitoneal reaction was studied experimentally by Kleinsasser and Warshaw, who injected barium mixed with stool into the peritoneal cavities of dogs, as well as barium alone and sterile stool. These authors concluded that neither barium alone nor sterile stool caused severe adhesions. Olnick and Watkins followed a patient who had barium enter the peritoneal cavity through a perforated ulcer 5 years before re-examination. There were no complaints referable to the barium. Radiographic studies showed barium in nodular deposits throughout the abdomen, including the space between the liver and the right diaphragm.

Rarely, spontaneous perforation of an undiseased pelvic colon occurs, perhaps incident to straining. Two such cases were reported by Henderson. In the first one very little peritoneal reaction was present because stool plugged the opening in the sigmoid, which was about 1½ inches in diameter. In the second patient, who also had a tear at the rectosigmoid, a diffuse peritonitis was present. Pneumoperitoneum likewise may follow proctosigmoidoscopy or cleansing enemas.

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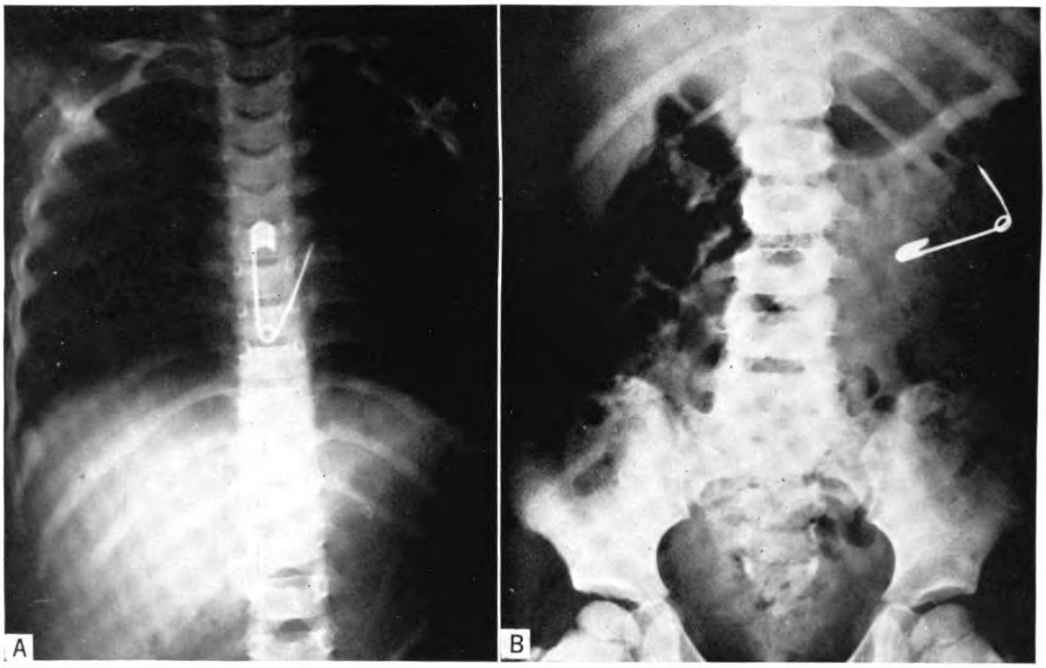

FIG. 215.—*A*, Open safety pin in the esophagus of a 2½-year-old child.
B, Same patient, 5 days later. Spontaneous passage and evacuation occurred.

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FOREIGN BODIES

The variety of foreign bodies which enter the gastrointestinal tract and pass through it seems to be limitless. We have observed patients who had swallowed objects as varied as pocket knives, safety pins, tacks, nails, bobby pins, toys, crayons, needles, dentures, toothpicks, and toothbrushes, just to mention a few. Infrequently, and usually under abnormal circumstances, strange objects appear in the colon, as for example a half-full bottle of Worcestershire sauce in one slightly intoxicated man who entered the emergency ward with the story that he had sat down hard on a table. Most of these can be identified on scout abdominal films. In most cases, relatively small foreign bodies can pass through the intestinal canal without ill effect. Nevertheless, observation is required to make sure that no obstruction or perforation takes place. The localization of foreign bodies in the upper abdomen is helped sometimes by the ingestion of a carbonated beverage. Gaseous distention of the stomach is useful in deciding whether an object lies within or outside the stomach. Magnetic removal of suitable foreign bodies such as pins from the stomach by means of an alnico magnet was first described by Silber, Epstein and Kaplan. An extensive

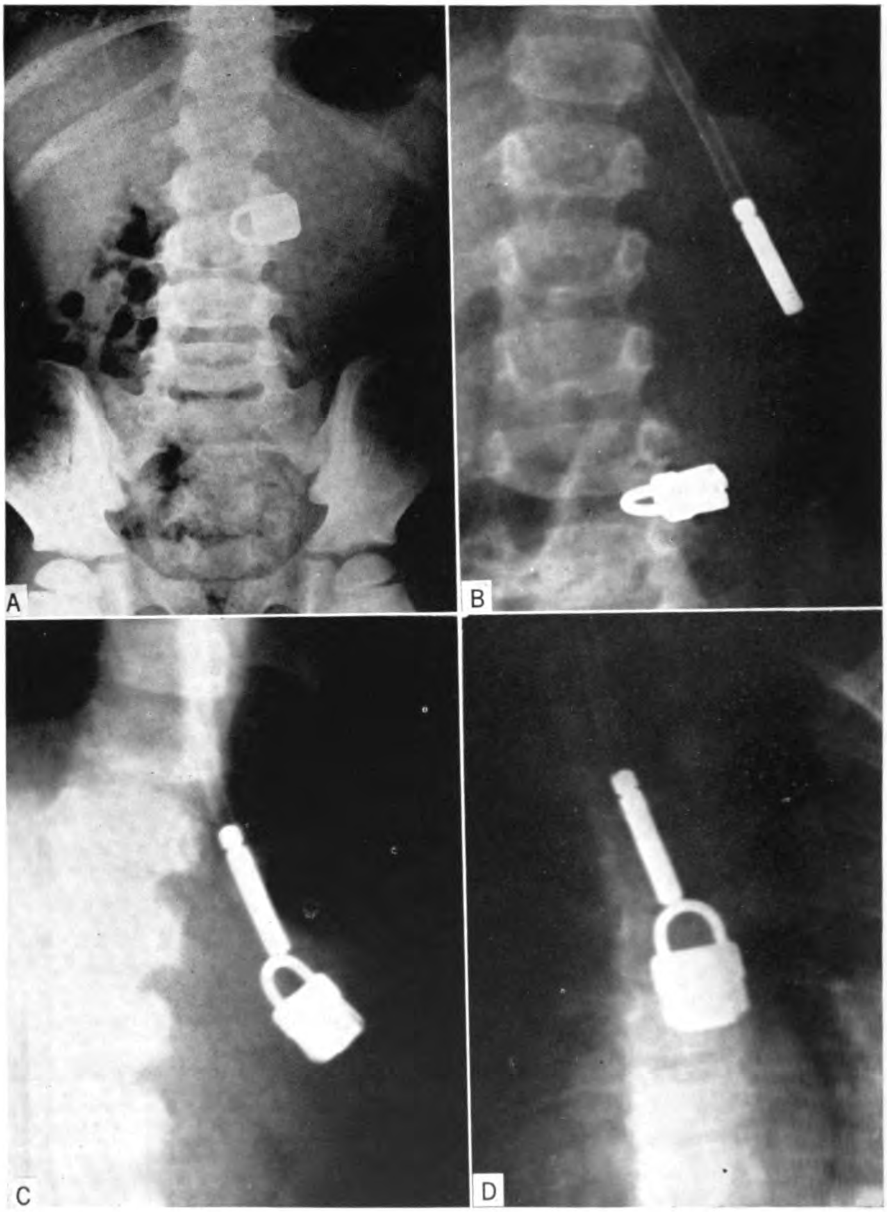

FIG. 216.—*A*, Removal of lock from stomach of a 5½-year-old boy. Abdominal film taken June 22, 1943, shows lock in stomach.

B, *C* and *D*, Successive stages of removing lock with alnico magnet.

series of such cases was recently described by Equen, who reported in detail on his experience with this technic.

Psychotic patients are known to swallow foreign bodies, and radiographic examination of their abdomens may disclose a tremendous variety of objects. These patients often have no untoward symptoms. However, with the development of pressure from such objects, ulceration, perforation or obstruction may follow. The usual places for obstruction to occur is at the pylorus, at the junction of the second and third portions of the duodenum, the ileocecal region and the

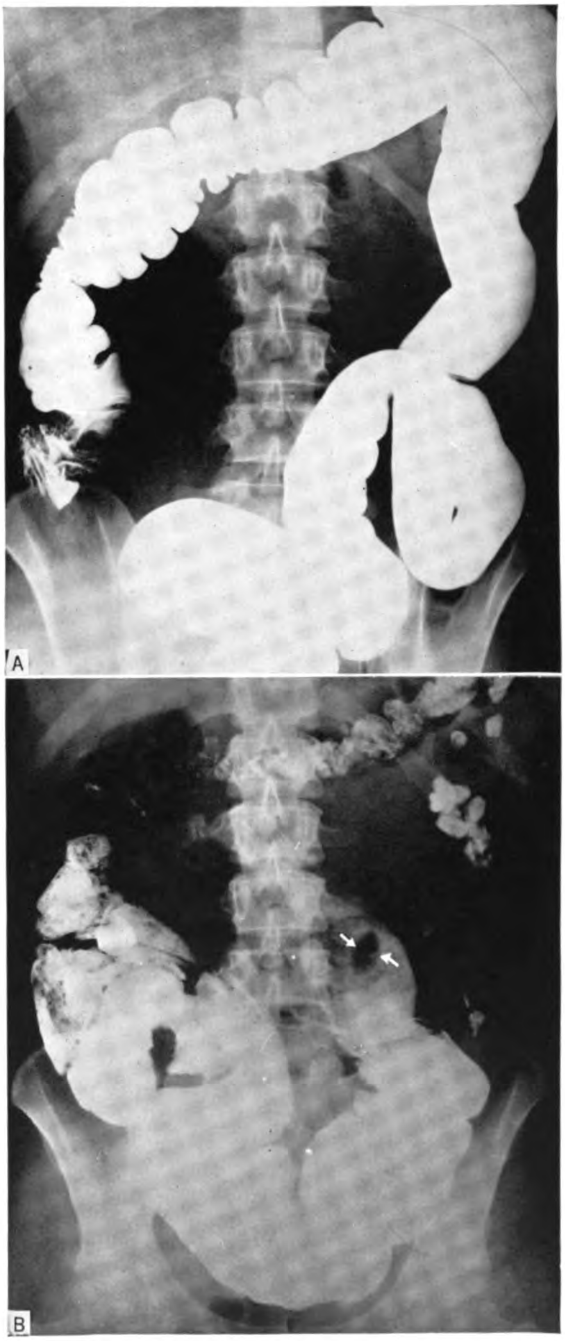

FIG. 217.—*A*, Barium enema of a patient operated for ruptured appendix 24 years before. Severely constipated. Abdominal pain and distention. Barium enema reveals defect at cecum, with gas-distended ileum adjacent thereto.

B, Same patient, 24 hours after barium meal. The ileum is markedly dilated and contains much foreign material. A fruit pit is clearly visible (arrows). At operation the terminal ileum and cecum were imbedded in dense granulomatous inflammation. Foreign bodies found in markedly dilated and atonic ileum.

appendix. Delay also may occur at the junction of the cecum and ascending colon and at the flexures and haustra of the large bowel.

The ingestion of radio-transparent foreign bodies is more serious. Perforation of the intestines by swallowed objects such as toothpicks can produce serious disturbances. Radiologic investigations in these patients usually disclose nothing to point to the presence of the offending foreign object, but may disclose some changes pointing to a local inflammation or granuloma. Occasionally, if the ingested object is sufficiently large it may be identified on a scout film.

Swallowed foreign bodies which do not pass through the intestinal tract sometimes assume rather bizarre appearances. We have seen patients in whom swallowed bobby pins remained in the stomach for several weeks. Because of the corrosive action of the stomach acid, the pins gradually disintegrated and finally were passed. The passage of a sharply-pointed object through the intestinal wall into the peritoneal cavity also may occur, and sometimes these reach remote places such as the omentum, the retroperitoneal areas, the mediastinum or even the extremities.

Every now and then various objects are left within the abdomen during surgery. Sometimes laparotomy pads may be identified by a telltale metal ring. Until the introduction of a radiopaque marker into sponges, these objects sometimes were permitted to remain within the abdomen and could not be detected until a granulomatous mass had formed about them.

The ingestion of material which disintegrates within the stomach may give rise to toxic symptoms, as occurred in a patient reported by Biehusen and Pulaski. This man suffered from lead poisoning after swallowing a key chain emblem containing lead. He was asymptomatic for about 45 days, and then developed anorexia, vomiting and weight loss. Surgical removal of the emblem on the sixty-eighth day relieved his symptoms. The lead poisoning was proved by stippling of the red blood cells and lead lines in his gums.

Occasionally the ingestion of fruit pits causes symptoms, particularly in young children. One of the hazards of this accident is aggravation of peptic ulcers in which the crater is invaded by the foreign body. In patients with encroachment on the lumen of the lesser bowel by extrinsic adhesions, ingestion of fruit pits may convert a relatively asymptomatic partial intestinal obstruction into one requiring surgical relief. Patients with poor dentition have a tendency to swallow large chunks of unchewed food, raising the possibility of esophageal or gastrointestinal obstruction because of impaction of bulky food masses.

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BEZOARS

By definition, bezoars are gradual accumulations of undigestible material which collect in the stomach gradually. If the offending substance is plant material such as persimmons, or fruit or vegetable fibers, seeds and skins, the condition is referred to as phytobezoar or hortobezoar. If hair is ingested, and this is the most common situation, the term applied is trichobezoar. A combination of the two is considered a trichophytobezoar. The ingestion of foreign materials such as shellac, asphalt or putty produces intragastric concretions. The eating of common dirt is designated as geophagia.

As a rule, bezoars produce large gastric defects, and sometimes are large enough to form a cast of a fairly distended gastric cavity. On plain films of the abdomen a radiolucent area with entwined radiopaque strands may be identified. When barium is swallowed, it flows around the mass and produces a reticular pattern following the contour of the stomach. As the barium soaks into the mass, the shadow persists for a relatively long time. With multiple bezoars, the barium fills the interspaces between them, demonstrating the individual masses. Impingement against the pyloric ring may produce obstruction with gastric dilatation.

The clinical symptoms produced by bezoars may be insignificant. However, when the mass becomes large enough it causes pain, vomiting, anemia, exhaustion and intolerance for food. Passage of a bezoar into the intestinal tract induces obstruction, sometimes associated with perforation of the bowel. The pressure of a bezoar also can cause ulceration of the adjacent mucosa with consequent bleeding.

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ENTEROLITHS AND BARIUM GRANULOMAS

Stasis of the fecal current in the large intestine may be followed by compaction of feces and the formation of enteroliths. This is fairly common in elderly patients who are chronically constipated, and has been observed with megacolon, stricture or inflammation of the bowel or neoplastic obstruction. Sometimes these concretions calcify. Analysis of such a stone by Harland showed it to consist of alternate layers of inspissated fecal and mineral matter. Occasionally a fecolith forms about ingested foreign material such as fruit stems or pits. While enteroliths are common in the large bowel, they also are found occasionally in the small intestine, particularly in patients with partial intestinal obstruction.

Fecalomas at times are completely asymptomatic. In elderly patients with poor intestinal activity, they may impact causing intestinal obstruction which in essence is due to a foreign body. Frequently the diagnosis is established by the demonstration of radiolucent fecal masses with distention of the proximal bowel. Usually barium traverses the bowel, but passage is slow and interrupted by the larger fecoliths. Appendiceal fecal concretions have been discussed before. The high incidence of acute appendicitis

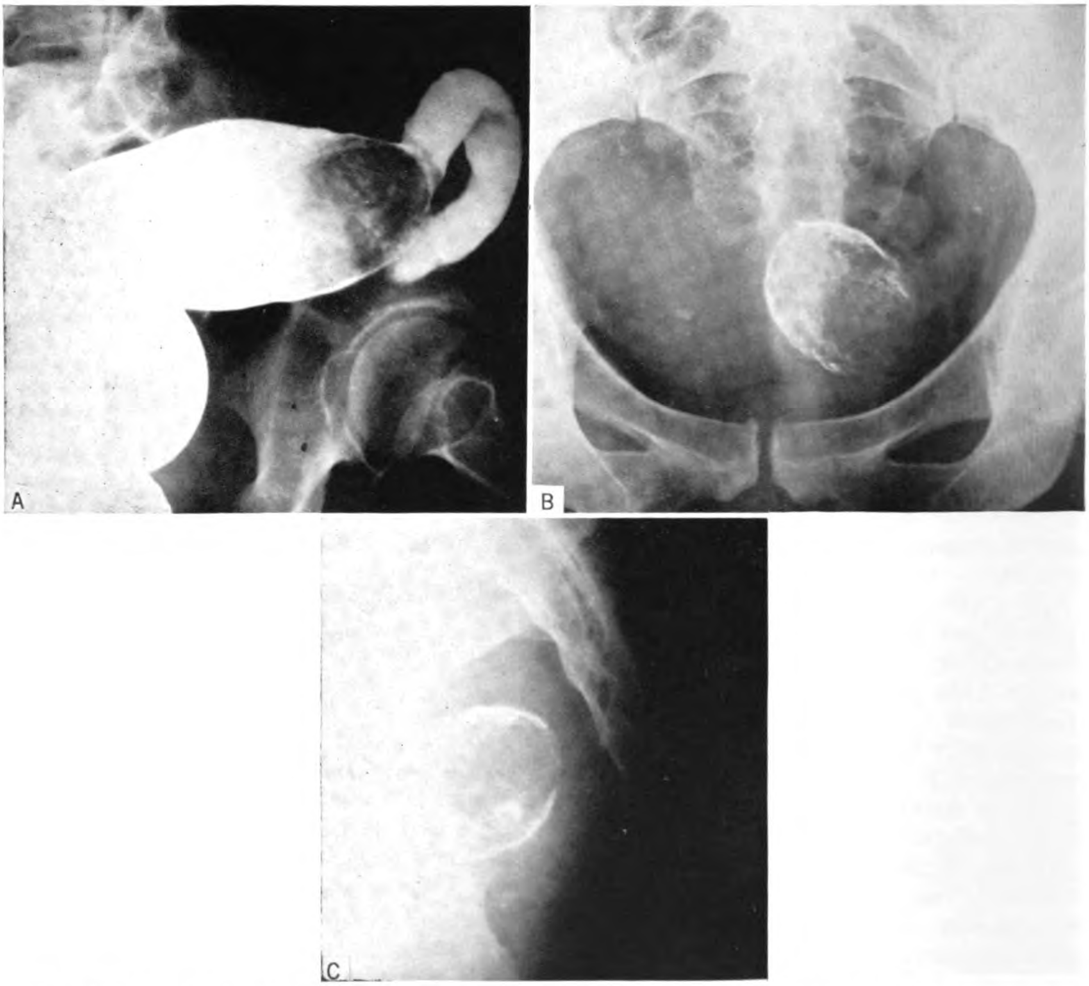

FIG. 218.—*A*, Obstruction at rectosigmoid due to large fecolith in a 51-year-old woman with constipation and pain.

B and *C*, Plain films made 2 weeks later show calcified fecolith in rectum. This was felt digitally and was removed with difficulty.

with perforation in the presence of such coproliths is deserving of re-emphasis.

The radiographic appearance of multiple fecoliths is quite characteristic on plain films of the abdomen. When these become impacted a considerable gaseous distention of the colon may appear. On barium enema examination there may be sufficient delay in the passage of the contrast material to represent an organic obstruction, particularly if the colon is atonic. Fecoliths caught in the splenic and sigmoid flexures are particularly prone to cause partial obstruction to the passage of barium. As a rule this occurs when multiple fecoliths are present, so that some indication as to the underlying cause may be had from the multiple negative shadows visible in the dilated colon. The wide variation in their bulk also is helpful. Some are but a centimeter or two in diameter, but others are over 10 cm. in diameter.

Gastrointestinal examinations sometimes are followed by the appearance of granulomas when some of the barium enters a small rent in the bowel, such as may be produced by trauma at the time of an enema.

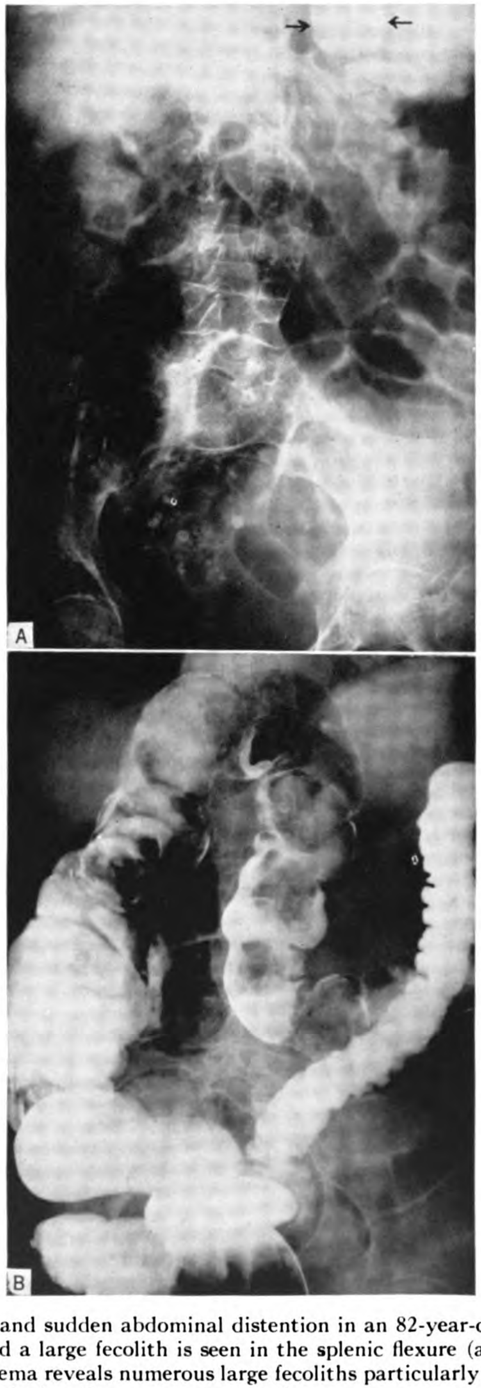

FIG. 219.—*A*, Constipation and sudden abdominal distention in an 82-year-old man. The colon contains considerable gas and stool, and a large fecolith is seen in the splenic flexure (arrows).

B, Same patient, barium enema reveals numerous large fecoliths particularly in ascending and transverse colon. Symptoms were relieved after these were evacuated.

If associated with an acute inflammatory reaction, there may be considerable local pain, as reported by Schwartz, and by Beddoe, Kay and Kaye. Barium entering the appendix may contribute to the development of a fecolith, but more often this is evacuated without injury or ill consequence. Enteric coated pills may persist for a relatively long time in the gastrointestinal tract, and can be recognized easily by their characteristic shapes.

Small intestinal obstruction infrequently may be caused by medications containing methylcellulose, or by curds of undigested protein milk.

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ARTERIOMESENTERIC OBSTRUCTION OF THE DUODENUM

Extrinsic duodenal obstruction occurs in the newborn period because of malrotation of the small intestine, with adhesions between the high cecum and the peritoneum in the subhepatic area which compress the third portion of the duodenum. Occasionally anomalous vessels originating from the root of the mesentery just above the pancreas extend into the duodenohepatic ligament and compress the anterior duodenal wall. In this way obstruction is produced which results in dilatation of the stomach and the proximal duodenum as well as the common bile duct and gallbladder. This condition is seen more often in adult females.

Chronic intermittent obstruction of the duodenum results from compression of the third portion of the duodenum at the point where it crosses the spine. When the superior mesenteric artery branches off in an unusually low position and is associated with visceroptosis, intermittent duodenal obstruction may follow. The possibility of intermittent arteriomesenteric obstruction of the duodenum is heightened in asthenic patients who have exaggerated lordosis and visceroptosis. It occurs most often in debilitated or emaciated people, particularly those who have been recumbent for a long time, and follows trauma or surgery in others. The obstructive process is intermittent, or may present with acute symptoms. The acute form, which has been observed in patients varying from 9 months to over 70 years old, is one of gastroduodenal dilatation with copious vomiting, nausea and periumbilical pain. The diagnosis is suggested radiologically by pronounced dilatation of the stomach and particularly of the first and second portions of the duodenum.

Chronic or intermittent arteriomesenteric duodenal obstruction is relatively more common. While it is attributed usually to compression by the mesenteric artery, it also is associated with congenital bands and malrotation of the intestine. Infiltration of the mesentery by malignant disease, pressure from enlarged lymph nodes due to tuberculosis or lymphoblastoma also may be cause for chronic duodenal ileus. The symptoms mainly are nausea, epigastric pain and occasional vomiting. Vasomotor disturbances and headache also appear. An association of chronic duodenal ileus with peptic ulcer has been encountered. The radiologic picture is one of varying intermittent gastroduodenal obstruction, with the point of

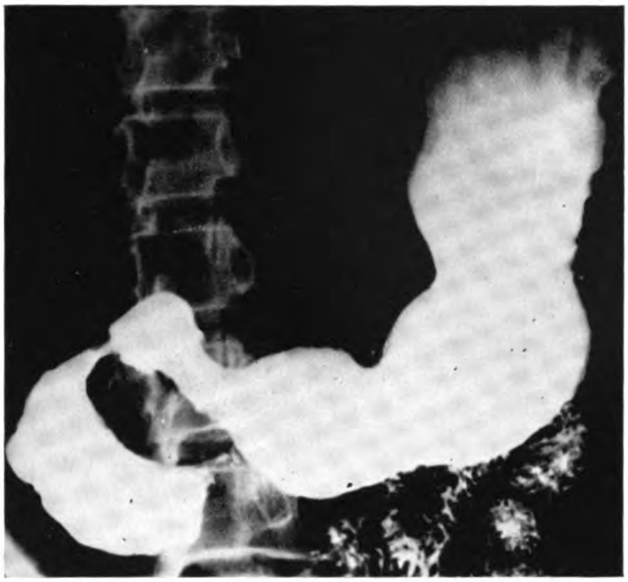

FIG. 220.—Arteriomesenteric obstruction of duodenum due to atypical superior mesenteric artery in a 29-year-old woman with weight loss and vomiting.

block represented as a relatively sharp linear marking. In some the duodenum reaches enormous size, and is associated with pronounced malnutrition. Reverse peristalsis in the duodenum often is prominent. Relief of this condition sometimes is obtained by changing the position of the patient and by gastric lavage. With reduction of the effects of asthenia and malnutrition, some relief also is noted. However, in certain instances nothing can be accomplished by medical treatment and surgery is required.

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CAST SYNDROME

Patients who are in heavy body casts sometimes develop acute gastric dilatation which may reach serious proportions, and has been associated with perforation and death. It has been suggested that the paralysis of the stomach musculature is the result of hyperextension of the spine and the considerably reduced excursions of the abdominal muscles. These lead to compression of the third portion of the duodenum by the mesentery and the superior mesenteric artery with consequent gastroduodenal distention. A tendency to swallow air aggravates this condition. Dorph collected 4 cases, 3 of which terminated fatally. His own case, an 18-year-old girl, had a fracture of the lower femur and was discharged after being placed in a body cast. Following this she began to vomit constantly, became alkalotic, and was readmitted in an unconscious state with the body cast still on. At postmortem examination acute dilatation of the stomach and duodenum up to the ligament of Treitz was found. No evidence of organic obstruction was present, the transition from dilated to normal bowel being abrupt. Rare though this may be, the demonstration of a dilated stomach in an immobilized patient should be regarded as a signal of danger.

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PNEUMATOSIS INTESTINALIS

The presence of gas cysts in the intestinal walls once was considered quite infrequent. Now, with radiologic help, the diagnosis is made more often. The condition occurs mainly in young people, and is found in children as well as in middle age. Males are affected more often than females. Parenthetically, the disturbance has long been known to exist in animals, particularly swine.

During infancy it has been associated with megacolon or other congenital obstructions of the intestinal tract. In children most of the gas collects in submucosal cysts, while in adults the gas accumulates in any layer of the bowel, especially in the subserosa. The cysts often are lined with a thin layer of endothelium. When the condition has been present for a long time giant cell formation and fibrosis occurs. At present it is believed that the gas enters the bowel wall on a mechanical basis. It is presumed that gas accumulates under pressure in the bowel lumen, and passes by way of a mucosal defect such as an ulcer to various areas along the mesenteric attachments. It then dissects along the subserosal layer, or follows blood vessels to the submucosa. The gaseous contents have been compared with atmospheric air, being odorless and non-inflammable, containing from 70 to 90 per cent of nitrogen and from 3 to 20 per cent of oxygen. The carbon dioxide content is from 0 to 15 per cent. These cysts vary in size from a millimeter or two to relatively large structure up to 8 or 10 cm. in diameter. Wolfe and Evans reported on the presence of gas within the portal veins of the liver in infants, demonstrated radiologically at postmortem as well as on premortem examinations. In 3 of their cases, pneumatosis of the bowel wall also was observed. It was believed that increased gaseous pressure within the bowel forced air into the mesenteric vein, which then reached the portal system. This observation supports the mechanical concept of the origin of pneumatosis intestinalis.

The distribution of the gas cysts is fairly uniform, occurring most often along the antimesenteric border of the ileal loops and the distal half of the colon. Less often the omentum, jejunum, duodenum and stomach are affected. When the colon alone is involved it is presumed that the condition followed some minute mucosal trauma. In cases of pneumatosis of the lesser bowel or the ileocecal area associated stenotic lesions may exist. Gaseous cysts also are associated with inflammatory processes such as enteritis, and have been related to infantile diarrhea. Koss described a form of emphysematous change in the bowel wall in children with ulcerative ileocolitis, with the gas distributed in the subserosa in sheet-like fashion. Gas cysts of the mesentery also appear, and occasionally the gas dissects into the retroperitoneal areas and accumulates between the diaphragm and the parietal peritoneum, giving a roentgenographic picture simulating pneumoperitoneum.

No specific symptomatology is associated with uncomplicated pneumatosis intestinalis. The condition usually is disclosed on radiographic examination made for complaints referable to a more basic condition such as congenital abnormalities, infectious, ulcerating or neoplastic lesions. In some patients ruptures of a cyst may result in pneumoperitoneum, which in rare instances has proven fatal. This condition also produces symptoms referable to the mechanical effects of the lesions, principally those of varying compression of the bowel lumen. With multiple cysts and an elongated mesentery recurrent volvulus may appear. Hemorrhage is a relatively infrequent complication.

Radiologically these cysts produce rather characteristic reticular double-ringed shadows in the wall of the bowel. Unusual collections of gas in the retroperitoneal area, or grape-like clusters of gaseous shadows around the periphery of the bowel suggest the diagnosis. When the condition is predominantly submucosal, more difficulty in diagnosis is encountered because the cyst walls overlap and merge with the intestinal lumen. Serosal gas cysts are manifested by extraluminal, smoothly outlined spherical or oval areas of diminished density. Skip areas of uninvolved bowel are frequent. Some dilatation of the lesser bowel with thickening of its wall also occurs together with reticular cyst-like multilocular translucent shadows.

On examination by barium enema colon involvement may be manifested by sharply defined impressions into the wall of the bowel secondary to pressure by the cysts. Soft tissue films may disclose the peripheral border of the cyst as well as the indentation produced against the barium column. On the post-evacuation and air contrast studies the cysts may simulate impressions secondary to distention of loops of bowel, but careful inspection of the periphery of the wall of the intestine should lead to the correct diagnosis. Separation of the liver from the right diaphragm by large emphysematous cysts has been described. In the differential diagnosis of this condition, the possibility of polypoid lesions, neoplasms and inflammatory diseases has to be excluded. Every effort should be made in patients with this condition not to overlook concomitant or underlying disease of greater clinical significance.

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RADIATION EFFECTS

In the planning of radiation therapy one must consider the results of the treatment on adjacent healthy tissues as well as its effect on the diseased area. It has long been known that the gastrointestinal tract is susceptible to injury from ionizing radiation. With the advent of increasingly potent forms of radiation, the matter requires serious reconsideration, particularly with high depth doses and relatively slight skin effects. Under these conditions the actual limiting factor becomes the gastrointestinal tract rather than the skin. Inasmuch as one cannot observe all of the bowel lumen during treatment, a precise knowledge of the effects of radiation on the deeper normal tissues is important in managing the patient. Help is available by proctosigmoidoscopic examinations, but information about the condition of the small intestine and the inaccessible areas of the bowel still is largely that estimated by experience.

Radiation therapy directed to the stomach in moderate doses results in a loss of activity of the glandular structures. Achlorhydria may appear, and it has been suggested that *x*-ray therapy directed to the stomach be used in the treatment of peptic ulcer. This is a rather drastic approach, and one which I would not recommend at present. The gastroscopic picture of post-irradiation gastritis was described by Palmer in 12 patients who had received million volt therapy for a variety of retroperitoneal tumors. He noted edema of the wall of the stomach with a tubular deformity and fixation of the antrum, and a fixed patulousness of the pylorus associated with deep chronic ulcers which healed without contractures. In 1 of the 12 cases he reported perforation and hemorrhage complicated the post-irradiation gastritis. Ricketts and his associates studied the stomachs of patients with peptic ulcers treated with *x*-radiation in doses of from 1100 to 2500 r, and noted redness and edema of the stomach mucosa with adherent exudate in 75 out of 77 patients examined. Changes appeared within a week to 3 months after treatment, and sometimes came at a time when the gastric acidity was reduced considerably. The irradiation gastritis was believed to be directly proportional to the decrease in gastric secretion. The inflammatory process was transitory, and was followed by atrophy. It was consistently present in patients with anacidity of 2 years or longer in duration. Patients in whom a nodular, cobblestone appearance of the mucous membrane suggested the presence of hypertrophic gastritis later had a normal or mildly atrophic mucosa. In this group the appearance of irradiation gastritis was not accompanied by adverse symptoms.

The small intestine appears more susceptible to radiation damage than the stomach. This may be due to functional disturbance of the lesser bowel which gives rise to alteration of absorption, with consequent dehydration, electrolyte imbalance and fluid loss from vomiting and diarrhea. The motility of the intestinal contents is increased during and immediately after irradiation and becomes depressed soon thereafter. A toxic picture may appear, with loss of body weight and malnutrition. The early irradiation changes of the small intestine include edema of the bowel wall, hyperemia, and friable mucous membrane. These appear during and after treatment. Mucosal ulcers appear in the ileum, and less often in the jejunum. These may be single, or multiple and confluent. The size of the ulcerations varies from minute to large shallow ulcers, which may become deeper and perforate the bowel. As a result of the mucosal irritation and ulceration, scarring and fibrosis develop. Ulcerations often are associated with a bloody diarrhea. In the event of perforation peritonitis and intra-abdominal fistulas occur. These changes also affect the mesentery with shortening and thickening of this structure. Lymphatic block may occur, which may produce chylous ascites. The later stages are characterized by atrophy of the mucosa, submucosal fibrosis, interstitial edema and hyaline thickening of the intima and media of the intestine. Lymphoid follicles may disappear and hyalinization of the muscle appears. Inasmuch as one of the profound effects of radiation is endarteritis, diminution of vascular supply may be followed by fibrosis, or at times the bowel wall becomes so devitalized that perforation occurs. As a result of the fibrotic changes intestinal obstruction which at first is partial and later becomes more severe is produced. The immediate effects of radiation therapy may persist for a month or longer after treatment has been concluded. The later effects often are manifested after a considerably longer time. It has been estimated that the tolerance of the small intestine is somewhere around 2500 r, using conventional apparatus.

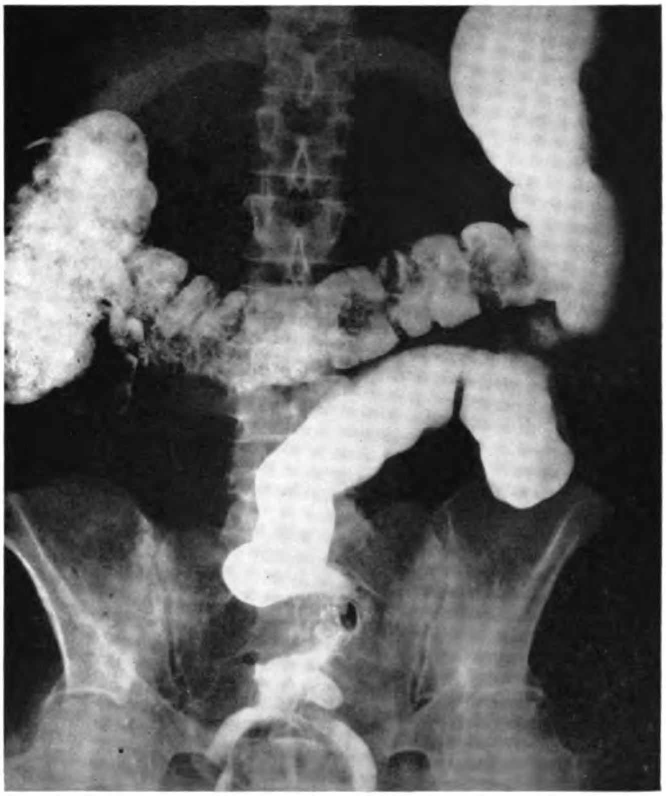

FIG. 221.—Proctitis after radiation therapy for carcinoma of the cervix in a 42-year-old woman. Proctoscopy revealed an edematous, reddened granular mucosa, which bled easily.

With supervoltage therapy, the maximum tolerance dose to the small intestine has been estimated at between 4000 and 5000 r. The end effects of radiation injury of the small bowel due to megavoltage therapy are more likely to be extensive fibrosis and perforation.

The colon is of approximately the same order of susceptibility to radiation damage as the small intestine. It is involved quite frequently, particularly in patients receiving pelvic treatment for cancer. Wide variation of individual susceptibility is seen, much the same as elsewhere in the gastrointestinal tract. The initial reaction is acute inflammation with bloody diarrhea, tenesmus and pain. Hyperemia and swelling of the rectal wall appears. As with the small intestine, the reaction may be delayed in its appearance and manifest itself 6 to 8 months after the conclusion of therapy. The presence of ulceration often causes relatively acute symptoms. As a result of the secondary endarteritis, gradual diminution of the blood supply may produce diffuse fibrosis which is difficult to distinguish from recurrent carcinoma. Radiation injury to the colon may be followed by obstruction, requiring surgical relief. As a result of contraction of the mesocolon there may be a shortening of the loops of bowel towards the mid-abdomen. Radiation injury of the pelvic walls and adjacent colon because of radium therapy is seen occasionally after treatment of carcinoma of the cervix. Increasing diarrhea with mucous and blood, occasional tenesmus and pain in the rectum on defecation appear soon after treatment. Proctoscopic examination of the colon discloses a kinked, edematous wall, a granular reaction of the mucosa with pinpoint hemorrhages and superficial ulcers. The contrast between the unaffected and injured wall is characteristically sharp. With recession of the acute reaction, the fibrotic changes are much the same as those incident to external irradiation. Perforation of the injured wall with vesicorectal or vaginorectal fistulas is a serious complication.

The radiologic demonstration of injury to the gastrointestinal tract secondary to irradiation is divided into the changes associated with acute injury and to the later stages characterized by fibrosis, scarring and loss of blood supply of the affected areas. In the acute stages, examination of the lesser bowel discloses delayed passage of barium through some of the loops, and hastened passage through others. Constriction of various segments with partial obstruction which may progress to complete block appear later as the fibrotic stage becomes manifest. Examination of the colon by barium enema during the acute stage discloses irritability and spasticity of the affected bowel, with deeply indented and irregular haustral markings. With fibrosis and scarring the bowel loops become narrowed, sometimes reaching the point of complete obstruction. Peristaltic activity of the intestinal loops is altered because of the fibrotic damage. Marked variation in the effect of radiation on the motor activity of the bowel is observed.

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CHEMICAL TRAUMA

The ingestion of corrosive chemicals causes both immediate and delayed gastrointestinal damage. Substances such as lye can produce pyloric obstruction secondary to sclerosis in a short time, if the patient survives. Two such cases were reported by Bolstad. Concomitant esophageal stricture was present in both. The ingestion of mineral acids such as hydrochloric or sulfuric acid can result in severe gastric damage with less change in the esophagus. A progressive type of gastric obstruction appears, associated with nausea, hematemesis and weight loss which reaches its maximum intensity after about 6 weeks. The stomach becomes thickened and scarred, particularly at the pyloric antrum, followed by obstruction which requires surgical relief. In some patients the irregularity of the stomach secondary to the effect of mineral acids simulates malignant disease. Hydrochloric acid is the most frequent offending agent, insofar as delayed reactions are concerned. Sulfuric acid is far more toxic and patients rarely survive long enough to develop fibrotic changes. However, should they live, strictures and irregularities simulating malignancy may appear.

The ingestion of strong alkalis produces considerably more esophageal than gastric change because of the neutralizing effects of gastric acid. Ferrous sulfate may cause a corrosive action on the stomach mucosa resulting in pyloric obstruction. This sometimes is observed in children who take an overdose of this medication. If the child survives, the acute gastric irritation is followed by contractures along the lesser curvature and pylorus.

In the past, and sometimes even at present, thorium dioxide is used for visualization of vascular structures or of the reticuloendothelial system. This substance is a practically inexhaustible source of radioactivity, emitting alpha, beta and gamma rays. Its extravasation into the soft tissues causes a severe granulomatous reaction. In addition to its local effects, later changes of a carcinogenic nature have been reported. Deposition of thorium dioxide in the liver may produce a diffuse sclerosis, with cirrhosis and severe degenerative changes. Similar alterations appear in the spleen, and the occurrence of neoplasms in the liver and spleen associated with thorium dioxide deposits have been recorded by Fruhling, Gross, Batzenschlager and Dorner.

Radiographically the deposition of thorium dioxide (Thorotrast) in the liver and spleen is recognized by the increased opacity of these organs. Occasionally other areas are affected. A case of deposition of Thorotrast in the wall of the gallbladder has been reported, the explanation being that interstitial hemorrhages within the gallbladder was followed by infiltration with macrophages, which then acted as part of the reticuloendothelial system.

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DRUG EFFECTS

A discussion of the toxic effects of the drugs in common use today would require far more space than is available here. However, because of the various effects of antibiotic, chemotherapeutic and various hormonal agents on the gastrointestinal tract, brief mention is worth while. Such changes often simulate organic disease, and it is important to take into consideration the factor of recent medication. In addition, the effect of drugs in the nature of altering the bacterial content of the bowel may give rise to changes which manifest themselves radiologically. An example of this is the effect of prolonged use of antibiotics permitting the emergence of fungi as pathogens, or the development of certain strains of micrococci as highly potent pathogens.

Several specific changes are worth reiterating. Among these are those associated with prolonged use of salicylates, which can

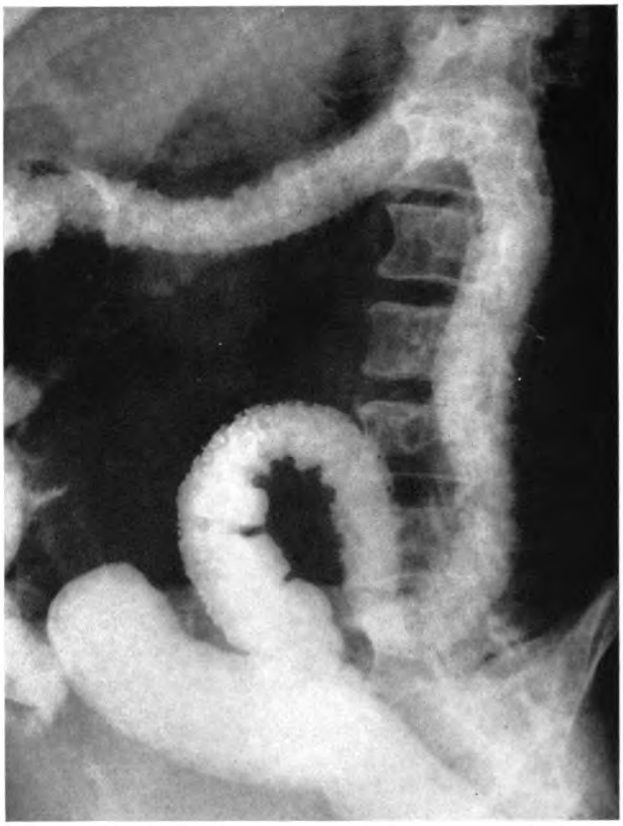

FIG. 222.—Colitis following phenylbutazone therapy. This subsided after therapy was stopped, and the bowel returned to normal in about 3 months. (Courtesy of Dr. M. Weiner.)

cause superficial ulcers associated with bleeding or reactivate preexisting ulcers. Phenylbutazone has come into considerable use for gout and other articular inflammatory diseases. The appearance of single or multiple gastric ulcers as a complication of phenylbutazone treatment is infrequent, but not rare. We have seen 4 such cases within the past 3 years. The first indication of this may be bleeding. Reactivation of preexistent ulcers also occur. The radiologic demonstration of peptic ulcers induced by these drugs may be difficult because of their shallowness.

Changes in the large intestine caused by treatment with tetracycline compounds have been reported, the clinical and radiological changes simulating those of ulcerative colitis. Similar changes are seen occasionally with phenylbutazone. Removal of the drug is followed in most cases by cessation of symptoms and return of the involved bowel to a normal appearance. Similar alterations occur with the sulfonamides.

Hexamethonium compounds used for the treatment of hypertensive disease may be accompanied by paralytic ileus, which sometimes is sufficiently intense to cause considerable distress. The use of chlorpromazine has been accompanied occasionally with cholangiolitis, giving rise to an intrahepatic type of jaundice attributed to a drug hypersensitivity reaction. Patients taking Banthine or Pro-Banthine sometimes have diminished peristalsis, with considerable gastrointestinal dilatation. As a rule, no definite symptoms are produced, but the stomach and duodenum in these patients may be so relaxed that adequate examination is difficult.

Patients who have undergone vagotomy

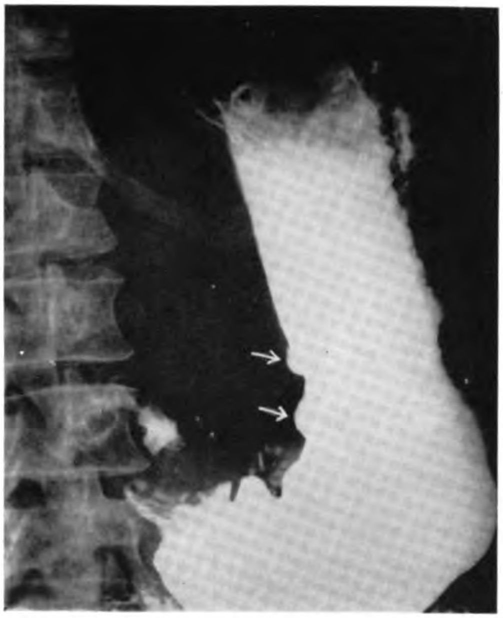

FIG. 223.—Double gastric ulcer (arrows) in a 43-year-old female with severe upper abdominal pain after being on phenylbutazone for 5 weeks.

sometimes have gastric dilatation and atony together with delayed transit time of barium through the lesser bowel.

The effects of various steroids on the gastrointestinal tract has aroused considerable interest. The activation of preexistent ulcers and the appearance of fresh ulcers is a possible complication of prolonged therapy. These patients are prone to develop large hemorrhagic ulcers which may perforate spontaneously, and which have very little in the way of clinical symptoms. The radiographic identification of steroid-produced ulcers often is difficult because of their shallow configurations and absence of fibrosis around the crater. Perforating ulcers are sometimes first brought to notice by the onset of pneumoperitoneum. As a rule, symptoms associated with steroid induced ulcers subside after treatment is stopped, but occasionally the complications require surgery.

Recently the association of inflammatory pancreatic lesions with steroid therapy has been mentioned. Carone and Liebow found postmortem evidence of acute pancreatitis or peripancreatic fat necrosis or both in 16 of 54 patients who had been so treated. Pathologic changes of ectasia of the pancreatic acini were seen in 59 per cent, as compared with 24 per cent of the control group. In a control group of similar age and with similar disease processes pancreatic changes were present in but two. The entire subject of drug-induced peptic ulcer was admirably reviewed recently by Kirsner.

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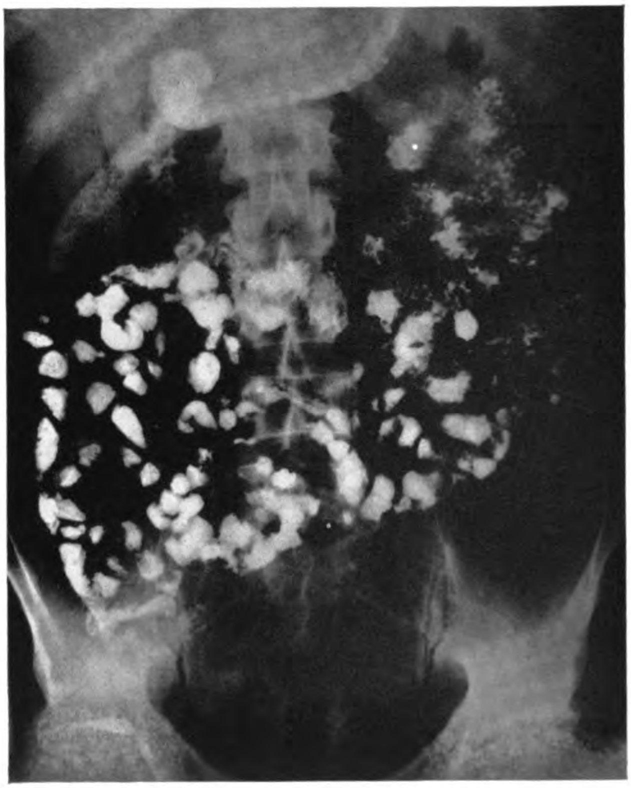

FIG. 224.—Stress pattern of lesser bowel irritability in a 42-year-old tense and excited male executive.

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EMOTIONAL TRAUMA AND DISORDERS OF THE CENTRAL NERVOUS SYSTEM

The close relationship between emotional stress and gastrointestinal disturbances has long been known. The radiologic demonstration of the effects of emotional tension has been studied in some detail lately. Emotional disturbances produced by an interview technic alters the mucosal pattern of the small intestine almost immediately from a normal one to a disordered motor function appearance. Friedman considered such an altered mucosal pattern as a functional variation within normal limits rather than a pathologic change. The presenting symptoms were not necessarily reproduced by the lesser bowel changes produced by deliberate emotional stimulation. Goin described a case of a Jewish woman, 54 years old, who had no ill effects from unknowingly ingesting pork. When this patient was first given barium and then offered pork she complained of violent abdominal pain, and striking changes in the small intestinal pattern were disclosed radiologically. Spasm of the small intestine sometimes reducing the bowel to a thin, string-like structure with intervening areas of dilatation may be seen in patients under emotional stress.

Ileus associated with colic, either urinary or biliary in nature, is well known, and is another manifestation of disturbed nervous impulses to the intestinal tract. Dilatation of the colon also appears under these conditions. We have seen several cases of marked spasm of the splenic flexure associated with left renal colic. In one patient the picture was that of an obstruction, believed to be due to a carcinoma. The true nature of the condition was clarified when a stone was found in the left ureter. The patient recovered rapidly after the stone was passed.

The association of ulcerations of the gastrointestinal tract, particularly of the stomach and duodenum, with central nervous system disorders was recognized by Cushing. These ulcers may be relatively acute and have no induration about them. They may be completely asymptomatic, and attention is directed to them only at postmortem examination or if they should perforate. The occurrence of similar ulceration in patients who had been severely burned was recently reviewed by Hummel, Lanchantin and Artz. These authors noted the appearance of Curling's ulcers in 20 out of a thousand burned patients. Of 80 who died, 17 had gastrointestinal ulcers, mainly in the duodenum. Several were multiple, and hemorrhage occurred in 10 cases. Only 3 patients had abdominal pain. The symptoms usually are mild, and the diagnosis of these may be delayed until operation or postmortem examination.

The occurrence of ileus with inflammatory lesions of the central nervous system is infrequent. Such a case was reported recently by Moffatt and Grant. We had the opportunity of observing a youngster with pronounced hydrocephalus who developed an ileus indistinguishable from intestinal obstruction. The patient expired and postmortem examination failed to disclose any lesion which could account for the ileus.

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GASTROINTESTINAL ALLERGY

As Ingelfinger, Lowell and Franklin pointed out, this is a diagnosis frequently considered, occasionally evaluated and rarely established. There appears to be no manifestation which can be regarded as definitely indicative of this condition. Radiologic studies which reveal patterns of atypical distention and contraction of the intestinal coils have been observed often, but this is so nonspecific that it cannot be regarded as more than suggestive.

Angioneurotic edema sometimes is associated with abdominal pain. Biering presented a case in which postmortem examination disclosed edema of the intestines from the duodenum to the colon of varying intensity in several areas, combined with pronounced eosinophilic infiltration. This patient had complained of intense abdominal pain, and on one occasion the radiographic studies suggested an intussusception. This was not verified at operation, and the possibility of this being transitory and spontaneously relieved was considered likely.

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